

Feb 7. 11

R39101

Digitized by the Internet Archive
in 2015

<https://archive.org/details/b21950507>

ON CONSUMPTION.

In preparation by the same Author.

ON DISEASES OF THE HEART
AND
THORACIC AORTA
INCLUDING
MEDIASTINAL TUMOURS.

ON CONSUMPTION

AND

ON CERTAIN DISEASES

OF THE

LUNGS AND PLEURA

BEING A SECOND EDITION REVISED AND EXTENDED OF
"THE VARIETIES OF PULMONARY CONSUMPTION."

BY

R. DOUGLAS POWELL, M.D. LOND.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, LONDON:
PHYSICIAN TO THE BROMPTON HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST;
LATE SENIOR ASSISTANT PHYSICIAN TO THE CHARING CROSS HOSPITAL,
AND LECTURER ON MATERIA MEDICA AT THE MEDICAL SCHOOL.

LONDON :

H. K. LEWIS, 136 GOWER STREET, W.C.

1878.

LONDON: PRINTED BY H. K. LEWIS,
136 GOWER STREET.

PREFACE.

THE work on the Varieties of Consumption, published by the Author six years ago, has now been for some time out of print, and in replacing it by one of somewhat wider range, he has felt reluctant to disconnect those chapters which have been already kindly received. The views there expressed were formed with much care from extensive clinical and post-mortem observations at the Brompton Hospital, and further experience has enabled the Author to find but little to alter in them. He is, moreover, assured that the clinical plan of grouping experience about single well marked cases, upon which the original volume was written, is better calculated to instruct than the more abstract method usually followed. It is indeed only by bearing well marked instances of disease strongly in mind, that the practitioner escapes becoming hampered and enfeebled rather than truly enriched in opinion and treatment, by his widening experience.

In the present work the subject of Consumption has been extended by the addition of chapters on Pulmonary Cavities, on Pneumothorax, on Tubercular Ulceration of the Bowels, and on Tubercular Meningitis, and a brief sketch has been given of the conditions upon which false or spurious Hæmoptysis commonly depends. The Author is aware that the chapter on the Physical Examination of the Chest is very brief and deficient in detail. It is, however, designedly so, for some experience in teaching has convinced him that the char-

acters and methods of eliciting Physical Signs can only be successfully taught at the bed-side. A systematic explanation of the mechanism and meanings of such signs would require much space, a profound knowledge of acoustics, and would be little read. A few practical hints, and a table of signs therefore, are all that has been attempted in the present work.

The remaining chapters on Bronchitis, Pneumonia, Emphysema, Pleuritis, and Pleuritic effusions still leave some important pulmonary affections unhandled, of which, however, cancer of the lungs and pleura will be treated of in association with tumours of the mediastinum in a separate volume, which will be devoted principally to diseases of the heart and great vessels within the chest.

Some final remarks have been added on the statics and dynamics of the chest, with the view of drawing greater attention to agencies of a physical kind, which the Author conceives to be more importantly concerned than is yet recognised in the pathology and symptoms of many pulmonary and cardiac diseases.

In conclusion the Author most gratefully acknowledges the kindness of his friend and colleague Dr. Reginald Thompson, for much valuable advice as the proof sheets have passed through his hands, and for the admirable coloured drawing illustrating tubercular laryngitis.

15 Henrietta Street, Cavendish Square.
March, 1878.

CONTENTS.

PART I.

CHAPTER I.

INTRODUCTORY REMARKS ON PULMONARY PHTHISIS	Page I
--	-----------

CHAPTER II.

PATHOLOGY.

Two kinds of morbid processes—Inflammation: affecting the parenchyma, Catarrhal Pneumonia in three degrees of intensity; affecting the fibrous stroma, Pulmonary Fibrosis—Tuberclle, nature and seat of, development—Hereditary predisposition to Tuberculosis and Consumption—Mechanical effects of the respiratory movements upon the Lungs and Pleura: pleural adhesions, mode of production, significance of friction—Thickening of pleura, how produced—Bronchiectasis	4
---	---

CHAPTER III.

THE PHYSICAL EXAMINATION OF THE CHEST.

Table of Physical Signs—Hints on Chest Examination	22
--	----

CHAPTER IV.

ALVEOLAR CATARRH AND CATARRHAL PNEUMONIC PHTHISIS.

Alveolar catarrh may advance to Catarrhal-Pneumonic, or Tuberculo-Pneumonic, or Tuberculo-Fibroid Phthisis; the first stage of phthisis most amenable to treatment—Diagnosis—Illustrative case of Catarrhal-pneumonic Phthisis in an early stage—Comments on ætiology, prognosis and treatment	26
--	----

	Page
CHAPTER V.	
Case of Catarrhal Pneumonia, non-hereditary—Significance of Fever and of the signs of softening; their relation to one another—Gradual change in Physical Signs; Supervention of Pulmonary Fibrosis—A second case briefly referred to—Further progress of such cases; a hint as to their management	38

CHAPTER VI.

Fibroid Phthisis, its characteristic signs and pathology summarised; reasons for selection of term as most applicable—A disease secondary to some parenchymatous affection of the lung; doubtful if pleurisy or bronchitis alone will give rise to it—Relations to other forms of phthisis—Example related—Summary—Remarks on diagnosis, prognosis, and treatment	46
---	----

CHAPTER VI.

On Hæmorrhagic Phthisis—Subject divided into Hæmorrhagic Phthisis proper and Recurrent Hæmoptysis; distinction between the two—True hæmorrhagic phthisis very rare; difficult to ascertain the true relation of the hæmoptysis to the primary disease: views of Niemeyer—Illustrative case of hæmorrhagic phthisis—Remarks: (1) Why considered a case of phthisis; (2) Why hæmorrhage not bronchial, and affecting lung secondarily; (3) Why probably coincident with and caused by active pulmonary congestion; general conclusion, with definition of Hæmorrhagic Phthisis; further points for and against hæmorrhage in such cases being bronchial—Disease has no relation to Hæmorrhagic Diathesis—Remarks on Treatment; value of thermometer as a guide	59
--	----

CHAPTER VII.

Recurrent Hæmoptysis: Illustrative Case—main features of the disease; repeated copious hæmoptysis; chronicity of pulmonary disease—Pathology; slowly forming or old excavation, not necessarily tubercular; frequent absence of secondary fever; modes of	
---	--

arrest of haemorrhage—Treatment, Prophylaxis—Both the forms of haemoptysis described rare; significance of true haemoptysis but little weakened by modern research.	69
---	----

CHAPTER VIII.

Alveolar Catarrh before spoken of may set up local tuberculisation; why disease tubercular, not simple chronic inflammation; seat; progress independent of pneumonia; how different from miliary tubercle; mode of extension by continuous growth; clinical differences—Chronic Tubercular Phthisis the best clinical name for this disease—Relationship of local tuberculisation to local, miliary, and general tuberculosis one of degree or intensity as regards infective origin or specific constitutional nature—Distinctive characters of Chronic Tubercular Phthisis; prognosis—Sketch of a case of acute Tubculo-pneumonic Phthisis; distinguishing characters from Acute Tuberculosis and Acute Pneumonic Phthisis—General rule as to prognosis; necessity of watching the signs of fever as well as physical signs—Table representing chief varieties of phthisis, with their distinguishing characters	80
--	----

CHAPTER IX.

LARYNGEAL PHTHISIS.

Laryngeal Phthisis, term used to signify those cases of Phthisis in which the Larynx is early or very prominently involved. Pathology of the disease—Morbid Anatomy. Symptoms and Signs. The use of the Laryngoscope. Diagnosis from Alcoholic or Irritative Catarrh, Syphilitic Laryngitis, Functional Aphony. Treatment	go
---	----

CHAPTER X.

ON PULMONARY CAVITIES.

Pulmonary cavities.—Why separately considered; their presence does not necessarily constitute the third stage of phthisis—Recent Cavities, their formation, physical signs indicating caseous abscess	
---	--

	Page
—Expectoration, significance of elastic tissue in expectoration the same as that of humid crackling râle: neither necessarily significant of present activity of disease—Extension of cavities—Rindfleisch's views—Symptoms: fever, chills, night sweats, expectoration—Treatment: quinine, arsenic, salicylic acid, salines—Inhalations and cough-mixtures rather to be avoided—Sputa should be expelled—Recent cavity may cicatrise, or become quiescent and contract: its bronchus may become obliterated—Secreting cavity, description of, signs and symptoms—Albuminoid degeneration of organs, diarrhoea—Objects for treatment. 1. To lessen secretion. 2. To evacuate what secretion is formed. 3. To disinfect—Counter-irritation, inhalations—Active or ulcerous cavity, description of, signs and symptoms—Treatment—Inhalations, support stimulants, ammonia and bark or iron, counter-irritation—Local treatment of cavities	100

CHAPTER XI.

PNEUMOTHORAX.

Pneumothorax—Laennec's views, controverted by Jaccoud—More commonly arises in course of phthisis than from other causes—May occur at any period—Mode of origin, (a) from sphaelus of pleura, (b) from fistulous perforation.—Composition of gas—Symptoms and signs vary with valvular or non-valvular nature of opening—Summary of symptoms. Physical signs—Hydro-pneumothorax—Percussion resonance, auscultatory phenomena, and cardiac displacement more fully considered—M. Gaide's views—Mechanism of displacement of heart. Case illustrative of—Dr. Hayden's views—Intra-thoracic pressure—Experiments showing effect of collapse and compression of lung upon circulation through it. Diagnosis of pneumothorax—Prognosis—Treatment	122
--	-----

CHAPTER XII.

ULCERATION OF THE BOWEL IN PHTHISIS.

Ulceration of the bowel in phthisis. Pathology unsettled. Morbid anatomy more definite. Appearances described. Seats of ulcer-

tion. Etiology. Symptoms vary somewhat with locality of ulceration. Rules for diagnosis. Chest signs and symptoms abate during diarrhoea, an important feature in diagnosis. Extensive ulceration may exist without diarrhoea. Caution needed in employment of purgatives in phthisis. Treatment. Mild cases. More severe cases. Importance of rest	144
---	-----

CHAPTER XIII.

TUBERCULAR MENINGITIS.

Tubercular meningitis,—most insidious in its supervention.—Illustrative cases exemplifying the most characteristic symptoms.—Comments.—Table of twenty cases showing prominent symptoms.—Conclusions from their treatment	154
---	-----

PART II.

CHAPTER I.

BRONCHITIS—BRONCHIAL CATARRH.

Definition—Etiology, age, sex, occupation, changes of temperature. “Catching cold,” mechanism of: dusty employment: exanthema: Influenza, hay asthma: dentition. Hereditary influence, heart diseases. Morbid appearances, a sample often seen during life in the trachea—Three stages of catarrh, congestive, transudative, secretory—Tendency of acute catarrh, (1) to recovery, (2) to become chronic, (3) to purulent inflammation, (4) to extension to deeper parts: 1 and 2 most common—Table summarising the history, distribution and tendencies of chief varieties of bronchitis—Symptoms of acute catarrh of larger tubes; physical signs—

	Page
Catarrh of smaller tubes, symptoms, physical signs—Diagnosis of bronchitis, from pulmonary oedema, tuberculosis and acute phthisis—Prognosis—Treatment of capillary bronchitis in the adult; in infants and old people—Treatment of chronic bronchitis—Secondary bronchitis not separately considered—Remarks on dust bronchitis and on the effect of inhaled irritants in causing phthisis: dust, cold and damp compared in their effects upon the bronchial tubes and lungs. Inhaled dust may produce catarrhal phthisis, chronic bronchitis with or without asthmatic paroxysms, or indurative phthisis—Illustrative case of dust bronchitis—Remarks	168

CHAPTER II.

REMARKS ON PULMONARY VESICULAR EMPHYSEMA.

Definition—Nature of defect in respiratory mechanism in emphysema—Conditions present in emphysema recounted, with comments—Etiology of the disease, partly mechanical; observations of Jenner, Gairdner, Rindfleisch and Greenhow referred to. Atrophic changes, precede and accompany the dilatation: earliest stage accounted for. Illustrative case of emphysema. Remarks on treatment	186
---	-----

CHAPTER III.

PNEUMONIA.

Pneumonia—a disease more familiar than common. Sketch of general symptoms of a case in mid-career: more detailed symptoms and signs.—Anatomical characters—Promontory symptoms, various in character and may suggest other affections than pneumonia—Signs of pneumonia continued. Defervescence. Resolution: occasional occurrence of secondary fever: explanation of, including <i>r��sum��</i> of morbid and restorative processes in pneumonia—Etiology—Definition of pneumonia—Prognosis and Treatment. Excessive temperature, abscess, gangrene and empyema, the chief dangerous events that may occur in pneumonia: their treatment	200
--	-----

CHAPTER IV.

ON DISEASES OF THE PLEURA.

- Pleuritis, varieties of—Simple pleurisy, causes: pathology: symptoms and signs: diagnosis; treatment—Local pleurisy, sometimes simple: generally from extension—Suppurative pleurisy: compared to purulent infiltration of the lung: symptoms: physical signs: difficulties in diagnosis: views of Bacelli—Pleuritic effusion, special signs indicating excessive effusion: position of the heart: axis of heart rarely transposed: illustrative case. Pulsation of fluid—Stress of circulation in opposite lung 211

CHAPTER V.

ON THE TREATMENT OF PLEURITIC EFFUSIONS.

- Simple inflammatory effusions: indications for paracentesis: methods of operating. Chronic Hydrothorax—Empyema: necessity of paracentesis: methods of operating, (1) antiseptic, (2) by single opening and injection of pleura, case related, (3) double opening and drainage—Chronic empyema, treatment of, case related 226

CHAPTER VI.

ON FALSE OR SPURIOUS HÆMOPTYSIS.

- Hæmorrhage may arise:—1, from the nasal membrane; 2, from ulcerated throat; 3, from intentional injury; 4, from the gums and alveoli; 5, from a scorbutic condition of the mucous membranes; 6, from *diapedesis* in anaemia; 7, from more copious mucous hæmorrhage in hæmophilia 265

CHAPTER VII.

THE RESPIRATORY MECHANISM IN HEALTH AND DISEASE.

- Mechanism by which first expansion of lungs effected—Elasticity of lungs not entirely relaxed at end of expiration—How lungs maintained in the semi-expanded state—Residual tension of lungs—Use of schema to illustrate mechanism of respiration—Explanation of conditions present in Emphysema, Pneumonia, Pleuritic Effusion and Pneumothorax, by means of the schema—Some disease effects of atmospheric pressure 272

LIST OF ILLUSTRATIONS.

	PAGE
1. Tubercular ulceration of the Larynx— <i>Chromo-lithograph</i>	xvi
2. Diagram of case of Fibroid Phthisis— <i>Woodcut</i>	50
3. Elastic tissue and fragment of small vessel from expectoration of patient with rapidly forming cavities— <i>Woodcut</i>	105
4. Elastic tissue and fragment of bronchus from expectoration of patient with active pulmonary softening— <i>Woodcut</i>	105
5. Temperature chart of active Phthisis treated by salicylate of soda— <i>Woodcut</i>	108
6. 2 Diagrams showing position of heart in case of pneumothorax— <i>Woodcuts</i>	132
7. Diagram illustrating experiment to ascertain the effect of collapse upon the circulation through the lungs— <i>Woodcut</i>	135
8. Tracings of thoracic movements in emphysema— <i>Woodcuts</i>	193
9. Diagram showing displacement of heart in extreme empyema— <i>Woodcut</i>	222
10. Trochar used with syphon in paracentesis— <i>Woodcut</i>	235
11. Fittings of Potain's aspirator— <i>Woodcut</i>	236
12. Tracings showing intra-thoracic pressure during paracentesis	264
13. Diagram of schema to illustrate mechanism of respiration	276



DESCRIPTION OF PLATE.

The plate is taken from the larynx of F. C. aged 22, whose voice became husky, in Jan., 1877, five months after chest symptoms had declared themselves. On admission into the Hospital in February, there was found extensive softening and excavation of the right lung and some softening at the left apex. The disease extended rapidly with febrile symptoms. There was from the time of admission complete aphonia, considerable pain and tenderness over the larynx, and much difficulty in expectorating. The nature of the disease was too obvious to require any detailed laryngoscopic examination, and indeed the pendulous epiglottis concealed the parts most extensively ulcerated. Sedative inhalations and repeated small blisters to the larynx considerably relieved suffering. The patient died one month after admission and seven months after the first onset of phthisical symptoms.

The plate (taken from a drawing by Dr. R. E. Thompson) shows the interior of the larynx seen from behind; the two halves of the thyroid cartilage, being drawn widely apart for this purpose.

The ulceration is seen to be situated chiefly above and below the cords and does not involve the upper part of the epiglottis. The mucous membrane over the lower half of the epiglottis is observed to be extensively eroded and the vocal cords thickened. The true cord on the left side is partly destroyed by the deep ulceration extending below it. The mucous membrane of the trachea is highly inflamed, and lines of ulceration are seen extending between the cartilaginous rings throughout the greater portion of the trachea. (See page 93).

ERRATA.

Page 14, lastline, for *disease* read *lesion*.

— 44, foot-note, for *last* read *22nd*.

Pages 81—89, Headings of pages, for *Alveolar Catarrh* read *Tubercular Phthisis*.

ON THE
VARIETIES OF PULMONARY PHTHISIS.

CHAPTER I.

INTRODUCTORY REMARKS.

MUCH as has been written upon the subject of Pulmonary Phthisis in all its aspects, it cannot be denied that there are still many questions concerning it upon which professional opinion remains undecided, and many of which are of the greatest importance. Let me mention some of them. How many kinds of Phthisis are there? Is there sufficient difference in prognosis between the several kinds to make it worth while to distinguish them? Are not all merely different degrees of the same disease or the same disease localised in different parts of the lung by different local causes? Does an attack of blood-spitting mean that the patient is already phthisical, or only that he is liable to become so as a result of the hæmoptysis? Is diarrhoea or laryngitis complicating a case of chronic lung disease a sure sign of the supervention of tuberculosis or is it not? Finally, and this question underlies all the others—it has excited more disputes than any of them—what constitutes Pulmonary Phthisis? Is it a perishable blood exudation into the lung, a blood disease, with, like many supposed blood diseases, a favourite seat for its anatomical manifestations? Or, is it the offspring of a mere catarrh affecting an organ so complexly loculated as to entangle and retain its own catarrhal products, so delicately sensitive as to become irritated to the point of ulceration by their presence, and, still further, so richly endowed with absorbents as to

readily take up and disseminate throughout its tissues, even to distant parts, the poisonous products of their decay?

Again, what part has the *constitution* to play in all this? We see a man stricken down with acute pneumonia, yet, let the disease be ever so severe, the lung ever so completely stuffed with exudation, we know that he will most probably, with no other treatment than judicious nursing, make a complete and perfect recovery. Another patient will be so trivially ill that he will scarce keep his room, and only on careful examination is it discovered that the lower part of one lung is solidified, yet this consolidation is far less likely ever to completely clear up, his lung may never be again sound. It may be that *constitution* is at the bottom of this difference, but it is I think more than doubtful whether it is the sole modifying cause. On the other hand, the difference of anatomical structure in the two cases is alone sufficiently apparent to account for the different results. In one, the first, we have a mere croupous exudation of fibrin and leucocytes, which can liquefy, resolve, and be absorbed with great facility as soon as the acute disease has passed. In catarrhal pneumonia, we have the air cells blocked with dense walled catarrhal cells, liquefaction and absorption of which is comparatively difficult, with but little accompanying fibrinous exudation to melt away into a molecular liquid and leave them free to escape. Let us glance at the catarrhal products distending any mucous follicle, *e.g.*, in the tonsils, or at the catarrhal collections occasionally found in the coecal appendix, or that sometimes remain stuffing a bronchus for years; they are rarely absorbed, but if not expelled inspissate and shrink, remaining as sequestered masses, perhaps at some later period to set up irritation, and escape by ulceration. But, granted that the difference between the two kinds of pneumonia above referred to is a structural one, is this structural difference but the anatomical expression of diverse constitutional states subjected to the same inflammatory test, or are the diseases in the two

cases distinct and interchangeable? We practically recognise the one as an acute sthenic, the other as a cachectic, pneumonia.

It is perhaps because consumption is still too much shunned, or watched with too little patience, at our large clinical hospitals, as being a disease of long duration and of ultimately fatal issue, in which the diagnosis once made, the prognosis is a matter of common inference, that the student in his after career, finds himself gradually arriving at a state of bewilderment, increasing with his growing experience, concerning the future of those who present themselves before him suffering from some one of its many forms. For one case comes before him with an amount of disease so slight that he cannot feel certain that any at all exists; the chest is only delicate, but the patient dies in a few months. He is shocked in another case to find a large cavity at one apex and his prognosis is very grave, yet the patient lives on in the enjoyment of fair health for years.

If, therefore, the few cases hereafter to be narrated illustrating some of the principal forms of Pulmonary Phthisis now recognised, with such practical comments on diagnosis, prognosis, and treatment as may be suggested by them, should tend in any degree, by bringing forward in an applied form the doctrines of the present day, to increase the precision and definiteness of the general knowledge of this always-prevalent disease, the object of this work will have been entirely achieved.

CHAPTER II.

PATHOLOGY.

Two kinds of morbid processes—Inflammation: affecting the parenchyma, Catarrhal Pneumonia in three degrees of intensity; affecting the fibrous stroma, Pulmonary Fibrosis—Tubercle, nature and seat of, development—Hereditary predisposition to Tuberculosis and Consumption—Mechanical effects of the respiratory movements upon the Lungs and Pleura: pleural adhesions, mode of production, significance of friction—Thickening of pleura, how produced—Bronchiectasis.

It is not proposed to enter with any historical or descriptive minuteness upon so wide a subject as the morbid anatomy of the forms of lung disease included under the common term Phthisis; but it would be scarcely possible to carry out the objects in view without first giving a brief sketch of those pathological changes upon which the clinical features of the different varieties of phthisis are based. It must here, too, be observed that, though for the moment the morbid processes going on in the lungs will be alone considered, yet this is in no disregard to the importance of constitutional changes of which we can take no anatomical note, nor of degenerations, or active complications of other organs, which are comparatively simple anatomically, and about which there has consequently been less confusion of terminology and vagueness of thought. To all these conditions reference can best be made incidentally in the discussion of the points specially illustrative of individual cases.

On inspecting the lungs of one who has died of phthisis, we meet with very great variety of appearances, which may, nevertheless, be recognised as the results of a comparatively few morbid processes; we see consolidation of the lung in every stage of formation, decay, and removal; and, glancing at the emaciated form before us, we have a very practical

definition of Phthisis Pulmonalis—progressive consolidation and decay of the lung with progressive wasting of the body.

As to the exact nature of the morbid processes which lead to this destruction of lung and waste of body, there are numerous and diverse opinions. These processes may, however, be said to be of two kinds. 1. Inflammation affecting with different degrees of intensity the different tissues of the lung, and running an acute, chronic, or chequered course. 2. A new growth—Tubercle—with its characteristic granulations disseminated through the lungs, or collected into nodular groups, or mingled with inflammatory changes, developing into fibroid tissue, or abruptly undergoing fatty change. We may meet with either of these processes in the acute or chronic form without any admixture of the other, but it is comparatively rare to meet with chronic tubercle unmixed with inflammatory changes.

It may be here recollected that fatty degeneration is the necessary consequence of inflammation, and is the means by which the products of inflammation become removable by absorption or expectoration, or sequestered for a time by caseation. The tendency of all new growth is also sooner or later to fatty decay, and in tubercle we find no exception to this rule.

There is yet a third set of processes of a physical kind, but having pathological results, which must be taken into account as leading to very important modifications in the signs of disease during life, and the appearances *post-mortem*—viz., 3. The respiratory movements of the chest walls, with the mechanical effects which they produce upon the lungs and pleuræ when modified in their physical conditions by disease.

(1.) The inflammatory process takes the largest and most important share in the production of the various appearances met with in phthisical lungs: it is the destroying element in this disease, as long ago pointed out by Addison.¹ It may, therefore be appropriately spoken of first.

¹ Collected writings published by New Sydenham Society, p. 56.

With that form of inflammation of the lung—acute sthenic pneumonia—agreeing in many of its characters with an acute specific disease (*e.g.*, idiopathic erysipelas), we have but little to do in dealing with cases of phthisis; we only meet with it as an exceptional complication. Doubtless, the subject of acute basic pneumonia already cachectic, or rendered so by neglect during the disease, may become phthisical; some cases of basic phthisis have this origin. But the pneumonia which is the most constant element of true phthisis, and which will indeed as already hinted be found the main factor in the more chronic and cachectic forms of basic lung disease, is of a very different kind; its onset is usually insidious, and its origin appears to be generally by extension of a catarrhal process from the finer bronchial tubes to the interior of the alveoli: hence its name—*catarrhal* or *broncho-pneumonia*. It is identical pathologically with the lobular pneumonia with which we are familiar in hooping-cough.² This form of pneumonia is essentially lobular, although the coalescence of many adjacent lobules may cause the consolidation of a whole lobe. The alveoli are affected by this inflammation with all degrees of intensity, from mere superficial catarrh causing slight epithelial desquamation to the most deeply destructive involvement of their walls.

² Professor Niemeyer states, on the authority of Bartels and Ziemssen, that pulmonary collapse always precedes catarrhal or broncho-pneumonia. This is a question of little importance to the present subject; but I cannot think that such is the case with the most typical forms of broncho-pneumonia. It certainly very often occurs in hooping-cough that inflammation succeeds to collapse, less frequently so in measles; but induration and agglutination of the air cells is the result—not their occupation by the large catarrhal cells characteristic of true broncho-pneumonia. Professor Niemeyer's description of broncho-pneumonia, as presenting, on cutting through the consolidation, a smooth surface, makes it appear that he must have drawn his description from collapsed lobules, which have become subsequently inflamed. In my experience, certainly, lobules of catarrhal pneumonia are strikingly granular on section.

In the simplest alveolar catarrh the cellular products may escape with the expectoration, leaving the alveolar wall undamaged. In the next degree of intensity, the alveoli and minute bronchi become blocked with the large granular cells, which are produced in great abundance. These cells, thus stuffing the alveoli, almost immediately begin to undergo fatty degeneration—the process by which resolution is naturally effected. They may liquefy, and be partially absorbed, partially expectorated; but the alveolar walls have been damaged, and permanent local collapse remains behind from their agglutination. This is the natural cure of the disease in this degree.

More commonly the cellular products, after having undergone complete fatty degeneration, become inspissated by absorption of fluid matter, and remain for a long time—perhaps for the lifetime of the patient—in the cheesy condition, or subsequently become cretaceous. This may be called natural arrest by obsolescence, and these cheesy masses are commonly looked upon as “old tubercle.”

In the still more intense degree of the process—catarrhal pneumonia—now under consideration, the alveolar wall is deeply involved in the inflammation, so that it subsequently undergoes fatty degeneration together with its cellular contents to an extent which varies with the intensity of the process, and breaks down in the subsequent liquefaction, gradually or rapidly according to circumstances.

The elastic tissue of the lung takes no active part in any of its inflammatory processes; it escapes but little altered when the alveoli break down, and thus, on being recognised in the sputa, affords certain evidence of pulmonary destruction. It will, of course, be understood that there is no real line of demarcation between the degrees of severity above described separately. The intensity of the first attack may at once determine the depth of injury, or the lighter may gradually pass into the graver degree.

But, in addition to the parenchyma^s proper of the lung, which, with its epithelium, is the special seat of catarrhal pneumonia, there is the fibrous stroma, if one may so style it, formed by the interlobular areolar tissue, supplying sheaths to the vessels and bronchi, contributing also to the formation of the alveoli, and intimately connected at the surface of the lung with the investing pleura. It could not be expected that an inflammation of the lung of any great severity would leave this widely spread tissue untouched; and it might also be anticipated, on reflection, that a tissue thus, comparatively speaking, deeply placed would, as a very general rule, only be affected secondarily to disease of the parenchyma or pleura. From this interstitial tissue are derived the tough, fibrous, pus-secreting walls of cavities, and the trabeculæ which for a long time resist the most severely destructive processes. The inflammatory process in this tissue is, as a rule, a much more deliberate one; even when in a state of active ulceration, as in the walls of some cavities, the destruction is molecular, sphacelus is rare. Inflammatory reaction in this tissue, indeed, more generally partakes of the character of growth under irritation, producing a more or less general condition of *fibrosis* of the lung. Decay, however, the inevitable result of inflammation, finally sets in; the fibrous tissue, at first merely hypertrophous, loses its characters as such; its nuclei, at first very abundant, gradually fade; its fibres fuse into tough homogeneous bands, and in their turn become granular and fatty and finally crumble away.

The various primary diseases—broncho-pneumonia, croupous pneumonia, chronic tubercle, pleurisy, etc.,—upon which

^s The term “parenchyma” is conveniently, and perhaps correctly, restricted to the minutest bronchi and the alveoli into which they are expanded. These minutest bronchi be it remembered have their lining membrane already so far modified as to cease to be strictly speaking a mucous membrane: the epithelium having become flattened and tessellated, and muciparous glands being no longer discoverable. (Stricker).

pulmonary fibrosis supervenes are thereby marked by clinical features of great interest and of significance for prognosis. But sometimes the fibrosis is so extensive as to become, whatever its origin may have been, the essential disease. Such cases have been very conveniently classified separately by Dr. Andrew Clark under the term "fibroid phthisis." He regards the disease as sometimes of idiopathic origin, or—what amounts to nearly the same thing—as a disease which progressively invades and destroys the lung from some one point of origin, as a local pleurisy or bronchitis. I must confess that in my much smaller experience I have not yet met with a case in which the fibrosis has appeared to me to have been either idiopathic or to have thus extended widely beyond the primary disease without the supervention of another disease, such as tubercle, or a repetition of the primary malady. I will refer to this again, however, later.

(2.) It would be impossible at the present time to gain a fair notion as to the essential nature and mode of origin of tubercle without first, however briefly, glancing at the recent anatomical discoveries which we largely owe to the labours of Dr. Sanderson and Dr. Klein⁴ respecting the distribution of the lymphatics in the lung. The full significance of the complicated lymphatic system of the lung thus disclosed cannot as yet be fully appreciated even in its physiological bearings, and it must be long ere we can fully ascertain the limits of its participation in pathological processes.

If we commence our scrutiny in the walls of the pulmonary alveoli we find that the spaces of the capillary vascular network contain, indeed constitute, lacunæ or lymph spaces which are lined or loosely occupied with protoplasmic connective tissue cells: the lacunæ intercommunicate by fine processes and discharge into lymphatic vessels which in the depth of the lung accompany the minutest subcapillary blood-vessels (perivascular lymphatics) but which on the surface of the

⁴ The Anatomy of the Lymphatic System, Part II. The Lung.

lung occupy the grooves between the lobules (sub-pleural lymphatics). The connective tissue cells lining the lacunæ in the alveolar walls send fine protoplasmic processes which insinuate between the epithelial cells of the alveolar surface (pseudostomata) and thus maintain the lymphatic system in living contact with the air spaces. The subpleural lymphatics inosculate by deep branches with the perivascular lymphatics and further communicate directly with the pleura by means of fine canals which open upon its surface (stomata). Both these sets of lymphatics course respectively onwards to the bronchial glands. The bronchial tubes also give rise to lymphatics (peribronchial lymphatics) which have their rootlets in fine canals and intercommunicating spaces in the mucosa in direct communication with the mucous surface by protoplasmic processes of connective tissue cells projected between the columnar epithelial cells. These bronchial rootlets discharge into the larger lymphatic vessels of the adventitia, which, like the rest course onwards towards the bronchial glands at the root of the lung. The perivascular and peribronchial lymphatics being adjacent freely inosculate especially at their finer divisions in the depth of the lung. Connected with the peribronchial lymphatics distinct acini or rudimentary glands have been discovered by Prof. Burdon Sanderson in the lung of the guinea pig, and it is reasonable to suppose that they are also represented in the human lung although their existence has not as yet been demonstrated. The whole lung is thus pervaded in every crevice of its structure by lymphatic tissue consisting of branched protoplasmic cells communicating with fine tubes and interstitial spaces occupied or lined with endothelial cells.

Sufficient evidence has now accumulated to render it tolerably certain that tubercle is a morbid growth of the lymphatic gland class, and that it may be a mere hyperplasia of the gland tissue normally minutely disseminated through the organs of the body (Sanderson).⁵ The characteristic form

⁵ For the development and further organisation of this lymphatic new

of tubercle is the grey granulation, a hard semi-transparent cartilaginous looking nodule of about the size of a pin head, intimately connected with the surrounding tissue. Each granulation consists essentially of a collection of small rounded lymphoid cells with a delicate reticulated stroma, the cells being derived by multiplication from the lymphatic elements of the lung. For the most part the granulations have no definite vascular supply although it has been pointed out by I think Dr. Bastian that a vascular zone sometimes surrounds young granulations.⁶ From their close contiguity to vessels, however, their nutritive supply is assured until from the dense multiplication of cells their central portions suffer deprivation. Whilst Dr. Burdon Sanderson has especially observed the sheaths of the minute bronchi as favourite sites for the origin of the tubercular granulations (there being, in the guinea pig at all events, minute gland acini in these situations) Dr. Wilson Fox has on the other hand pointed out a remarkable obstruction to the minute vessels of the lung as most characteristic of the tuberculising process, and Dr. Klein has shown that the ultimate branches of the pulmonary artery become both invaded by the growing adenoid tissue from without and thickened and obstructed at points by proliferation of the endothelial lining of their own inner coat. The alveolar walls, the peribronchial, perivascular and subpleural tissues, are then the seats of tubercular growths: but under special circumstances of irritation tubercle may be developed upon the surface of the pleura from the endothelial cells of the stomata, and it is quite conceivable that this growth may similarly intrude into the alveolar spaces from the protoplasmic processes constituting the pseudostomata which, it will be remembered, are parts of the lymphatic system. It is obvious growth is required "no more than the presence at the seat of the irritation of a pre-existing element of the group of connective tissue corpuscles and endothelium." Buhl quoted by Klein *op. cit.* p. 69.

⁶ *Vide* also plate 23 of Klein's work.

however that broadly speaking *the seat of tubercle is the interstitial connective tissue of the lung.*

The anatomical relationship of tubercle to Phthisis and to tuberculosis respectively is a question upon which very conflicting opinions have been expressed. Until the time of Addison and indeed up to the researches of Virchow upon tubercle, Laennec's views held good, and the tendency has again been of late to give no inconsiderable support to the doctrine of this long abused pathologist, who regarded tubercle as the anatomical lesion of Phthisis and its caseation and softening as the cause of the lung destruction in that disease. It was not long after Bayle's and Laennec's teachings became known before the "tubercle corpuscle" was discovered, and now again the revival of the views of the great French pathologist receives dubious support from the recent discovery of the "giant cell" regarded by some as the criterion of tubercle. This newly found disease-product has however not met with a uniformly cordial reception. Dr. Wilson Fox doubts its existence as a cell, having failed to isolate it from the surrounding tissue; its source and composition have been most differently explained. It seems most likely to result from a mere fusion of young cells with their contained nuclei.⁷ Before being aware of the existence of this peculiar "cell" I had repeatedly seen in sections of grey granulations of some date and becoming horny, an appearance identical with it which I regarded as due to commencing fibroid organisation of tubercle. It is probable however, that this "mother cell" may be produced in several ways and it is certain that tubercle may exist without it.

Seeing that the lymphatic elements from which tubercle admittedly grows everywhere pervade the lung, it is easy to conceive that we may have tubercle not only in the form of

⁷ Hering believes these "cells" to consist of the coagulated protoplasmic contents of lymphatic vessels entangling lymph corpuscles. Klein however speaks of them as true cells and states that they can be isolated.—*op. cit.* p. 77 also plate 27.

isolated nodules (miliary tubercle), but also in a more diffused form thickening alveoli and consolidating the lung by more or less extensive tracts of adenoid tissue (diffused tubercle).

It is impossible on anatomical grounds logically to refuse this admission, and it is in this more liberal acceptation of the term tubercle by Dr. Wilson Fox, that one step on the return to Laennec's view may be regarded as taken. Let us, however, in this connection carefully note the difference between the terms *tubercle* and *tuberculosis*. Tuberculosis is a disease-process of the definite nature of which we are daily becoming better informed, tubercle is a disease-product. The process tuberculosis may be said to consist in the more or less wide or universal dissemination through the body of a poison-influence which results in an outbreak through the infected region of innumerable tubercle growths. But tubercle may arise locally by direct infective irritation and may thence occur not in isolated granulations but in the more diffused form.

Hence there need be no ambiguity about the term tuberculosis which signifies an acute and more or less general outbreak of miliary tubercle, whilst tubercle itself, which in the form of the miliary granulations is the essential lesion of tuberculosis, may yet arise locally, outside as it were, the general system.

Under favourable circumstances⁸ tubercle undergoes development into a peculiar form of fibroid tissue, at first very recognisable from ordinary hyperplastic fibrous tissue, but which subsequently becomes converted into bands or tracts of uniform homogeneous texture, and finally fattily degenerates and is removed. This development of tubercle, before its final decay, has hardly been sufficiently insisted upon as an essential character always observable if circumstances permit the attainment of the necessary stage. It is, however,

⁸ The death and removal of tubercle may be greatly hastened by associated inflammatory processes.

in strict accordance with the lymphatic gland type of this morbid growth, and it is of some importance as affecting the clinical characters of chronic tuberculosis. In acute tuberculosis, the patient does not often live long enough for any process of the kind to take place. In chronic pulmonary tuberculisation, however, and when tubercle attacks a lung rendered quiescent by previous disease, the stages of development of tubercle into fibroid tissue may be seen.

Having said this, however, it is necessary to remark that many distinguished pathologists in this country and abroad refuse to admit the adenoid structure of tubercle, affirming that the true fibrillated stroma, with stellate cells in the angles of its network, always seen in the follicles of a normal lymphatic gland, is not to be recognised in tubercle. The stroma of tubercle differs from that of a gland follicle, according to these observers, in that it is made up of homogeneous fibrils shewing no trace of cellular development, and, it is hinted, that this stroma in tubercle is altogether a product of the hardening process to which the specimen has been submitted and is not to be recognised at all in fresh sections. It must of course, however, be remembered that the structure of tubercle is regarded as analogous to that of lymph-gland tissue indurated and increased by irritation—under which circumstances the cells become densely packed, and the gland stroma loses its cellular structure, and, finally, assumes the tougher homogeneous form indistinguishable from that of tubercle. I cannot help myself being firmly persuaded that this view of the lymphatic nature of tubercle is far more in accord with the results of careful microscopic observation than that which would class it amongst the ordinary products of inflammation.

The tendency of modern research—the experiments on inoculation in animals, and the very powerful advocacy of the late Professor Niemeyer—is certainly to show that tubercle is much more commonly a secondary disease than has until

lately been suspected—that people are, in fact, only very exceptionally, if ever, born to die of tuberculosis. A due appreciation of this doctrine, so different from that even now accepted by many, is of almost national importance in giving encouragement to those hygienic and other measures of prevention, the neglect of which has too often been sanctioned by a foregone conclusion. It would, I think, however, be extremely injudicious to deny hereditary predisposition to tubercle altogether. Moreover, when we come to the question of hereditary predisposition to those forms of consumption which originate in catarrhal pneumonia, it is freely admitted that the offspring of consumptive parents have a tendency to this form of pulmonary phthisis, that the scrofulous have a like tendency [Niemeyer], and that scrofulosis is sometimes hereditary.⁹ Again, from the tendency to the occurrence of chronic inflammation, especially in glands, leaving behind cheesy deposits, by which the scrofulous diathesis is characterised, it is regarded as the constitutional state in which true tuberculosis is most likely to occur. My own observation would not enable me to agree in this latter view.

These statements necessitate a considerable addition to the list of those who are hereditarily liable to tubercle in the old-fashioned sense of the term. But it must be remarked that catarrhal pneumonia and scrofulosis can be more efficiently guarded against, by attention to climate, soil, etc., and more successfully treated, than truly tubercular disease. My colleague, Dr. C. Theodore Williams, the latest authority on the question of hereditary predisposition to consumption (in its broad sense), in a paper read before the Medical and Chirurgical Society in January, 1871, gave 48 per cent. as the proportion in which, out of 1000 carefully noted cases, family predisposition could be traced, using the term “family” to include brothers and sisters and first cousins. It thus appears that, even making every allowance for alterations in terms

⁹ Waldenburg, *Die Tuberculose*, etc., p. 524.

and views, fewer people die of hereditary consumption now than formerly. It may be, of course, that traditional opinion has simply been erroneous in regarding consumption as so strongly hereditary, but it is perhaps nearer the truth to say that under the influence of superior hygienic circumstances, since Sanitary Science has been so much popularised, hereditary predisposition, as strong and real as ever, gets fewer opportunities of being nursed by neglectful hygiene into confirmed disease.

To return from this digression—perhaps a pardonable one, considering the importance of the subject—there are yet a few words to be said respecting the mechanical effects of the respiratory movements of the chest walls upon the lungs and their investing pleura.

(3). In health, ordinary inspiration is a muscular act, by which the elasticity of the lungs is overcome, and their expansion is effected to a certain degree. Ordinary expiration, on the other hand, is the elastic recoil of the lungs bearing with it the chest parietes to a point beyond that to which their natural resilience would bring them. There is, consequently, a resilient force in reserve, which goes to help muscular action in the first part of inspiration.¹⁰ It is easy to see how greatly this arrangement adds to the smoothness of the mechanism of respiration, the commencement of each act of which is normally almost wholly accomplished by elastic power. A glance at one diseased condition—emphysema—in which the elasticity of the lungs is impaired so that they do not contract to the normal extent in expiration, awakens our attention to the importance of what may at first sight appear a trivial matter: the thoracic parietes in emphysema are not drawn in beyond their position of rest—*i.e.* that position which they would naturally attain on a free opening being made into the pleural cavity. The abrupt and jerking manner in

¹⁰ For further development of this view see a paper “On some effects of lung elasticity in health and disease.”—*Med. Chir. Trans.*, 1876.

which inspiration commences in cases of emphysema, being effected by a conscious, albeit an habitual, effort on the part of the sufferer, cannot be regarded without offence to one's instinctive sense of mechanical perfection. But in cases of Phthisis we have to do with local, rather than general, alterations in the pulmonary texture; diminution, rather than enlargement, of the space occupied by the lungs; increased local resistance to expansion, rather than diminished tendency to recoil; and the parts within the chest which are most affected by these causes are the pleura and the bronchial tubes.

Why is it that pleural adhesions are so common in chronic lung diseases, and especially in phthisis? The answer usually given is—Firstly, that inflammation of the lung is very apt to extend to the pleura; secondly, that tubercle is very prone to attack serous membranes, and tubercular pleurisy is conspicuously a dry adhesive pleurisy. Both these statements are doubtless true, so far as they go; but they are by no means sufficient to explain the frequent and inevitable pleurisies and local adhesions of Phthisis, and notably of those varieties of phthisis about which there is most dispute as to their having anything to do with tubercle.

Confining our attention now to cases of Phthisis, though these observations are really applicable to other chronic pulmonary diseases, there are a few points worthy of remark concerning pleural adhesions.

1. They are pretty accurately limited to those portions of pleura corresponding with diseased lung beneath.
2. The more contractile the lung disease, and the tougher its texture, the thicker the pleura covering it.
3. *Post-mortem* there is frequently to be found no tubercle at all in the adhesions, and still more frequently no tubercular granulations in the pleura immediately in the neighbourhood of the adhesions.
4. In cases of very chronic phthisis (not secondary to pleu-

risy), with contraction and induration of part of one lung, we find *post-mortem* opposite the oldest part—*e.g.*, the apex—the two pleural layers perhaps intimately fused together, forming a white fibrous covering half or three-quarters of an inch in thickness. Lower down, however, we find the layers, each somewhat thickened, separated by a striated jelly-like material—œdematous connective tissue—the fine striæ of which pass vertically from one pleural surface to the other.

The real explanation of the recurring pleuritic pains and adhesions in cases of phthisis is, that when a portion of lung becomes damaged in texture by disease it ceases to follow accurately the expansile movements of the chest-wall; a certain gliding or rubbing motion takes place between the two normally corresponding pleural layers at this point;¹¹ friction, local pleuritis, and adhesion result. We can readily understand, therefore, how it is that a friction sound is often the first evidence we get of local pulmonary disease, and that a new friction sound means most generally more than mere dry pleurisy; it means, in fact, an accession of lung disease.

When the lung disease is of a very chronic indurative, contractile character, as in the cases referred to above in section 4, the effect of the continued inspiratory efforts to expand the toughened lung is to stretch out the adhesions and to separate the pleural layers to a certain extent; the further contraction of the lung continues the process, so that the parietal and visceral pleuræ may become separated by a considerable interval of half or three-quarters of an inch. This space is at first filled by serous fluid effused into the meshes of the areo-

¹¹ Most authors state that a gliding movement naturally takes place between the two pleural surfaces. I am myself inclined to think that in perfect health no such motion occurs, but that the expanding lung accurately, follows the expansion of the chest. Whether this correspondence in expansion be perfectly accurate or no, however, the gliding and friction must be greatly exaggerated over the region of a diseased portion of lung, and the explanation of adhesions in the text remains good.

lar tissue of the stretched adhesions. We thus get the œdematous pleura. At a subsequent stage, however, of the disease, by the continued growth of the areolar tissue, the whole space becomes occupied by tough fibrous tissue, and the two layers become completely welded together into one uniform fibrous thickness. That this is the real history of the enormous thickening of the pleura in many cases of chronic phthisis I have satisfied myself by repeated observation.¹²

It has seemed to me that thickening of the pleura has been regarded too much in the light of a dangerous pathological process, liable to extend into and by its contractile power to squeeze out of existence, so to speak, the proper lung tissue, whereas it will be found on careful examination to be most generally a condition secondary and quite subsidiary to the lung disease.

In primary pleuritis the thickened pleura is produced in a different way. After absorption of the fluid a certain thickness of lymph often remains between the two layers of the pleura, into which the granulations from each surface penetrate, and finally unite, completing the adhesion. There are many cases of phthisis of the pneumonic kind of tolerably acute progress, and attended with little contraction, in which, though the pleural surfaces are inflamed and covered with finely granular lymph, they do not become united. It is in these cases that pneumo-thorax is especially likely to occur.

Bronchial dilatation (bronchiectasis) is another important morbid condition with which the chest movements have something to do. The main causes of bronchiectasis may be summed up as follows:—1. Damage to the texture of the bronchial tube—atrophy of mucous membrane and thickening of fibrous coat (Biermer).¹³ 2. Increased air-pressure during cough, acting principally at those portions of the lungs where

¹² Vide *Path. Trans.*, vol. xx., pp. 59-61.

¹³ *Krankheiten der Bronchien und des Lungen-Parenchyms: Handbuch der speziellen Pathologie.* Virchow.

there is least support, notably the apices. 3. The expansion of the chest wall, or rather the struggle to expand it, failing to affect the air cells which are obliterated by disease, acting indirectly upon the bronchial tubes. 4. The diseased lung in its contractile forms, contracting in various directions tending to widen the imbedded bronchial tubes.

The constant movement of the lungs no doubt goes far to modify and hasten the progress of morbid processes going on within them. This must have occurred to the minds of many Physicians, and even more forcibly to Surgeons, who have to deal with wounds of the lung. We certainly often see in the lung compressed by fluid or air, a complete arrest of the phthisical process which had been previously active there. At the same time it must be remembered there is also in such cases complete and often permanent arrest of the function of that lung. Had we complete command over the duration of any effusion into the pleura we might artificially induce, the possibility of employing such a line of treatment might be entertained. At present the question is worthy of being raised whether in the acute stage of one-sided apex disease any method for restraining the movements of that apex, might be adopted, as *e.g.*, by binding the arm to the side, to this point, however, I shall refer in a future chapter. The constant admission of air to the softening tissues of the lung is also a condition impossible to remove, but which must be borne in mind in regarding the peculiarly destructive character of the inflammatory processes affecting it.

In fine I would wish, at the risk of some repetition to draw especial attention to the peculiarly intricate construction of the lung as well as to its constant movements by which much of the inveteracy of pulmonary diseases may be explained. Let us think of the lung roughly as an intricately infolded membranous surface, closely allied to and continuous with the bronchial mucous membrane, but peculiarly rich in bloodvessels and lymphatics.

tics. The products of a catarrh affecting the larger bronchial tubes can be readily expelled without difficulty or danger, but a much greater difficulty attends the removal of the products of a similar affection of the alveoli. Indeed, it is accomplished only in small part by expectoration, those portions which are not readily liquefied and absorbed are apt to accumulate, and by their subsequent decay to irritate the alveolar wall and to set up in it those proliferative and inflammatory changes which constitute local "tubercle"—just as the retained secretions of a sebaceous follicle may give rise to the acne pustule. The thickened alveolar walls in their turn degenerate and soften or suppurate, and in these few changes occurring in numerous centres corresponding to the lobular groups of alveoli, we find the very basis and foundation of the chief morbid results of Phthisis. Add to this group of changes the definite growth, miliary tubercle, which may arise in the neighbourhood infected from the caseous foci, or which may spread throughout the system from such centres;—in a word, add a description of what we understand by the term *tuberculosis* to the Phthisical changes we have enumerated, and all the active processes which produce pulmonary destruction in nine out of ten cases of consumption are summed up. The ceaseless movements of the lungs and the constant access of air to the diseased surfaces, keep up the suppuration and maintain the tendency to the absorption of septic matters.

I have now passed in brief, and I fear very imperfect review some of the main points in the pathology of phthisis which have a direct clinical bearing, and a due appreciation of which will, I think, materially assist in the correct reading of the cases as they appear before us in the subsequent chapters.

CHAPTER III.

THE PHYSICAL EXAMINATION OF THE CHEST.

TABLE OF PHYSICAL SIGNS—HINTS ON CHEST EXAMINATION.

THE relationship between physical signs and the diagnosis of pulmonary disease—is of a strictly practical kind, although of course depending upon acoustic principles, and he who would become a successful auscultator *i.e.*, a good diagnostician, must study auscultation in association with morbid anatomy. His mind will then gain at the bed-side through the stethoscope in most cases an accurate picture of the lung or heart or pleura under examination as though the organ were exposed to his view. This intimate association in the mind between physical signs and the lesions which give rise to them is only to be acquired by genuine clinical and *post-mortem* observation, no amount of reading or clinical work alone will suffice for its attainment.

I do not propose, then, to enter upon any detailed explanation of the auscultation and percussion signs that may be elicited in different kinds, and degrees of chest disease. Such an exposition will be found in Dr. Walshe's classical work or in that of Dr. Gee on *Auscultation and Percussion*. But with the permission of my colleague Dr. Cotton I have taken from his book on Phthisis a table which contains a “Vocabulary of physical signs, sufficient for every practical purpose.”¹⁴ To this table I have ventured to add a third column, giving what I think will be generally conceded to be the most usual significance of each of the signs named, and thus serving in the practical sense above alluded to, to define the meaning of the terms used.

¹⁴ *Phthisis and the Stethoscope*. 4th edit., p. 7.

TERMS.	SYNOMYS.	SIGNIFICANCE.	
Percussion Sounds ...	{ Clearness ... Dulness ... Wooden Character ... Tympanitic resonance ... Amphoric resonance ... Cracked-pot sound ... Weak ... Harsh ... Exaggerated ... Suppressed ... Jerking ... Expiratory murmur prolonged ... Bronchial ... Cavernous ... Amphoric ... Sonorous ... Sibilant ... Crepitant ... Subcrepitant ... Rhonchi ... Vocal Resonance... Cough Sounds ... Laryngeal Sounds ...	Normal ... Hardness Dry crackling ... Humid crackling ... Mucous ... Cavernous ... Bronchophony ... Pectoriloquy ... Ægophony ... Amphoric resonance ... { Bronchial ... Cavernous ... Amphoric ... Metallic tinkling ... Thoracic fluctuation ... Friction murmurs ... Harsh laryngeal respiration ... Sonorous rhonchi ... Sibilant rhonchi ... Gurgling ...	Health. (Needs confirmation by other signs). More or less consolidation, or lung displaced by fluid effusion or tumour. Thickened pleura, lung induration. Air in the pleura, or greatly dilated lung. Empty rigid walled cavity near the surface widely communicating with bronchi. Deficient movement of tidal air, diminished function. Undue dryness of tissue or partial consolidation. Increased movement of tidal air, increased function. Lung distant from surface, or bronchus obstructed. Irregular expansion, partial consolidation. May be nervous. Partial consolidation. Consolidation. Large cavity. Partially obstructed bronchus, bronchitis larger tubes. Ditto, smaller bronchi; bronchitis smaller tubes; bronchial spasm. Pneumonia. Thin fluid in bronchial tubes with consolidation around. Softening "tubercle" 1st stage (heard at end of inspiration), Softening "tubercle" 2nd stage (heard with inspiration and expiration) Secretion in bronchial tubes without consolidation around. Fluid secretion in a cavity. Voice conducted through a solid lung. Voice intensified by a cavity. Voice conducted through a compressed lung and a thin layer of fluid. Large echoing cavity with small opening; pneumothorax. Narrowed bronchus. Cavity near the surface containing some secretion. Large dry cavity—pneumothorax. Sound of a drop of fluid echoing through large cavity—pneumothorax. Air and fluid in large cavity—hydro-pneumothorax. Roughened pleural surface—pleurisy. Larynx partially obstructed by thickening or paralysis of chords. Secretion about laryngeal opening. Spasm of larynx. Much secretion with want of power to expectorate.

The commonly accepted regions of the chest are *anteriorly*, the supra clavicular, clavicular, infra clavicular mammary infra mammary on the right and left sides respectively: and the supra sternal, upper sternal, and lower sternal in the *median line*: *laterally* are the axillary and infra axillary: and *posteriorly* the upper scapular (supra spinous region), the lower scapular, the infra scapular, and the inter-scapular regions respectively on the two sides.

In physically exploring a chest, a general inspection of its surface should be first made to note any flattening or want of symmetry on the two sides. The movements of the two sides of the chest should be compared during both calm and deep breathing and the position of the heart's apex should next invariably be noted. Percussion should then be employed, each region of the chest being compared and the boundaries of any dulness or morbid resonance being noted not only in a downward direction but in a horizontal direction also. The encroachment of dulness upon the median line or the extension of the resonance of a lung across the median line are points of great importance both in diagnosis and prognosis. Auscultation follows in the same general order; the boundaries or superficial extension of each type of morbid sound being ascertained.

The sitting posture is the best if the patient be not in bed for examination, and whilst the back is being examined the patient should be directed to lean slightly forward and to let the arms fall loosely downwards between the knees, in which position the supra scapular regions at which the very summits of the lungs are found are best exposed for percussion and auscultation. Care must be taken not to be misled by noises made in the mouth of the patient when breathing, such fallacies will be avoided by directing him to open the mouth or to cough, or by instructing him, if necessary, how to breathe. An examination of any doubtful spot should never be completed without trying a *tussive* in-

spiration *i.e.*, the patient should be directed to cough while the stethoscope is still applied and auscultation carefully made during the inspiration preceding, and at the moment of, cough, also during a deep inspiration immediately after cough. At either of these moments a crepitant sound may be developed which would otherwise have quite escaped notice.

One remark more on the matter of percussion. It matters little¹⁵ what kind of instrument is used for auscultation but in percussion the fingers should (unless for some special reason or for the purpose of demonstration to a class) always be used in preference to Piorry's pleximeter. Not only can we usually rely upon carrying our fingers about with us, but the sense of touch most importantly supplements that of hearing whilst percussing the chest. With the gentlest percussion it is quite easy to *feel* qualities of dulness or hardness which the ear alone cannot detect. Fluid vibrations may be thus sometimes noted or a *bruit de pot fêlé* so called, detected by the finger, whilst to render the latter sound appreciable by the ear may require a force of percussion which is not justified by the importance of the result. As a rule much more information is gained by gentle than by forcible percussion although at times a somewhat hard stroke may be necessary.

¹⁵ Individual auscultators will have a preference for one form of stethoscope over another. For accuracy of conduction I believe that there is no instrument equal to the cedar stethoscope with a large slightly concave ear piece. The important point is, however, for each observer to keep to the instrument he has been most accustomed to use.

CHAPTER IV.

ALVEOLAR CATARRH AND CATARRHAL PNEUMONIC
PHTHISIS.

Alveolar catarrh may advance to Catarrhal-Pneumonic, or Tuberculo-Pneumonic, or Tuberculo-Fibroid Phthisis; the first stage of phthisis most amenable to treatment—Diagnosis—Illustrative case of Catarrhal-pneumonic Phthisis in an early stage—Comments on ætiology, prognosis, and treatment.

It would hardly be instructive to give an illustrative case of Alveolar Catarrh in the first and slightest degree, that stage which forms the connecting link between the prodromal catarrh of Niemeyer and catarrhal pneumonia. This condition is, however, an extremely common one, and may be very readily overlooked, for the signs by which it is recognised are only faintly marked. It must be considered as really the first stage of phthisis—through which, at least, all cases of catarrhal-pneumonic phthisis, and therefore the majority of cases of pulmonary consumption, pass.

The pathology of this disease consists, as has been before intimated, in the proliferation of the epithelium of the air cells by a catarrhal process of the most superficial kind affecting them. Tuberle has nothing whatever to do with this process, which may, indeed, pass on to catarrhal pneumonia and destruction of the lung without tubercle taking any conspicuous part in it. But an irritative over-growth of the minutely dispersed adenoid or gland tissue of the lung, which has been before referred to, may be set up secondarily by the catarrh, and then the disease in its further stages assumes the characters of that described by Addison as tuberculo-pneumonic phthisis. Further, the adenoid growth, accompanied, as it always is, by more or less increase of fibrous tissue, may,

having once been set going by the irritation of a simple catarrh, take so prominent a part in the future progress of the disease as to eclipse altogether the catarrhal process, and we get a somewhat rare, insidiously progressive and very destructive disease, which, I think, has not been fully described in its entirety, but which closely corresponds anatomically with the iron-grey induration of Addison, and invades the lung by substitution of a fibroid tissue, inch by inch, from apex to base.

These may be said to be the three directions in which alveolar catarrh when it runs an unfavourable course may lead, and its tendency to develope into such formidable morbid processes is a sufficient reason for its being naturally regarded as the first stage of phthisis. Hereditary predisposition, existing cachexia, and the special nature of the influences which have produced the disease play an important part in determining its future characters—*i.e.*, whether it shall be pneumonic, tuberculo-pneumonic, or tuberculo-fibroid; also, whether its course shall be rapid, or insidious, or intermittent. But I must hasten to observe that if alveolar catarrh be early recognised and rationally treated its progress may be in a large number of cases entirely checked, and the more adverse the circumstances which have led to the development of the disease, the more hope is there, on their removal, of convalescence.

How, then, can this condition be recognised in its earliest stage? Professor Niemeyer, who has more than any other author urged the importance of its early detection and treatment, regards the presence of pyrexia as the one symptom above all others indicating the extension of the catarrh to the alveoli, and he attaches some weight to streaky haemoptysis as a sign of the alveoli having become involved in the catarrhal process. The significance of these symptoms when present is beyond question; indeed, I think, when there is elevation of temperature we always have some pneumonia present, and have therefore arrived at a stage beyond mere catarrh.

The patient who is the subject of alveolar catarrh has always been depressed in health, through tardy convalescence from some other disease, or bad living, or mental anxiety: he has had a persistent, though it may be, a slight cough, for a longer or shorter time, and has during that time been getting thinner. At this stage the physical signs are very slight, but sufficient for diagnosis in conjunction with the symptoms and history. There is no dulness or impaired movement, but the respiration is weaker at one apex, the inspiration being wavy, or even jerking. There are usually a few sonorous *râles* present, which, if limited to that apex, are very significant; and, in addition, one hears at the extreme summit of the lung (supra-clavicular or supra-spinous region) a peculiar crumpling sound at the moment of cough, which differs both in time and degree from the crepitant sound audible at a somewhat later stage with the first inspiration following a cough.

Not to refine too much, though we have to deal with very slight physical signs—and, the slighter they are the more important are they to be recognised—we may say that physical signs of bronchial catarrh limited to one apex, and associated with a decided imperfection of the respiratory murmur at that apex, afford, when taken in conjunction with the symptoms—more particularly emaciation—unmistakable evidence of incipient phthisical disease, upon which we must advise most decidedly if we do not wish to see the patient pass beyond our control so far as positive cure is concerned.

There is no clinical line of demarcation to be drawn between this condition and the prodromal catarrh which precedes it, or the catarrhal pneumonia into which it is apt to pass; they shade imperceptibly into one another. I only specially refer to this stage as the earliest recognisable stage of phthisis. I now pass on to give a case illustrative of catarrhal pneumonia in an early stage, which also bears out some of the above remarks respecting the most common ætiology of this form of consumption.

Case I.—S. S., aged 29, a married woman engaged in domestic duties and suckling a child aged 7 weeks, came under my notice as an out-patient in April 1871. Her mother had died of consumption within two years of the patient's birth, and an elder sister had been affected with the disease in an early stage. She had enjoyed fair health until her first confinement, when she was with difficulty delivered of twins, only one of whom survived the birth, which was stated to have been a "cross one." This child she suckled for eleven months, when she again became pregnant with her present child. Ever since her last confinement she had suffered with increasing debility, emaciation, and cough, and shortly before that time she had had slight hæmoptysis.

She was a tall, thin, anæmic woman, with the worn look so characteristic of over-lactation or rapid child-bearing; her large, heavy, pendulous breasts, marbled with large veins, increased by contrast the apparent general flatness and narrowed antero-posterior diameter of the chest. There was no local flattening, however, at either apex, and the respiratory movements though generally deficient were not more so at one apex than at the other. On percussion over the summit of the left lung the resonance was somewhat less than on the opposite side; the respiratory sounds there, were harsh, and accompanied by some moist crepitatⁱon, which extended to the second rib. The respiratory murmur elsewhere was of fairly good quality, but somewhat feeble. The main symptoms complained of were troublesome cough and yellow expectoration, shortness of breath, general weakness, and giddiness in the head. The pulse was quick and weak, the appetite indifferent, but digestion fairly good.

The case was regarded as one of Catarrhal-pneumonic Phthisis in an early stage, the disease being limited to the left apex, and supervening upon the exhausting effects of more than thirty months' continuance alternately of gestation and lactation. She was directed immediately and completely to

wean the child, to take abundance of appropriate food, with a moderate amount of beer. Some counter-irritation was applied at the left apex, and cod-liver oil and steel wine administered, with some morphia and ipecacuanha lozenges for the cough.

On again examining the chest a month later the moist sounds were no longer audible with ordinary respiration, but a few crackles were heard after cough. There was slight flattening at the left apex, which became more obvious on deep inspiration; the respiratory sounds were feeble there, while on the opposite side they were more developed, and on percussion the line of resonance of the right lung extended a little to the left of the mid-sternal line. There was no evidence of a cavity at the left apex, and no extension of the disease below. The health of the woman was, though improved, by no means restored; she was still anaemic and thin. Her cough was troublesome, especially in the morning, and expectoration difficult, the effects of coughing often causing vomiting at that time. She had neglected to completely wean her child, and continued to "give it the breast now and then." The pulse was quiet but weak; the appetite improved. She had been unable to take a stronger preparation of iron ordered a fortnight previously, and was obliged to fall back upon the steel wine. Three weeks later she had very greatly improved in health and strength; some colour had returned to the cheeks, and she was gaining flesh rapidly.

This case, though not complete, illustrates so many points in the clinical history of catarrhal-pneumonic phthisis¹⁶ in its earlier stages that I have related it in preference to others.

I employ the term "catarrhal-pneumonic phthisis" because the term pneumonic phthisis is often used to signify cases which have commenced as basic pneumonia in which the

¹⁶ This case would be grouped under one of the following headings by the authors named:—Pneumonic phthisis, Addison; catarrhal pneumonia, Niemeyer, Herard and Cornil; epithelial pneumonia, Andrew Clark.

consolidation has not undergone complete resolution. It is true that we sometimes meet with genuine croupous pneumonia at one apex. I believe such cases are, however, exceedingly rare. The case now under consideration could not be confounded with such; the dulness was never absolute, the respiration was not decidedly bronchial,¹⁷ nor was the crepitation that of typical pneumonia.

But in other cases, which do not essentially differ from this save in degree, the dulness may be complete and the crepitation undistinguishable from that of true exudative or croupous pneumonia, the respiration being also tubular, but, I think, never so intensely so as that of basic pneumonia. This of course might be readily accounted for by the seat of the disease; but the subsequent course of such cases is very rarely indeed that of simple pneumonia. They do not undergo complete resolution. The most common course is for the consolidation subsequently to soften and break up into cavities; and on *post-mortem* inspection of such apex consolidations, we find the alveoli stuffed with large catarrhal cells, instead of being occupied by the fibrinous exudation with entangled corpuscles characteristic of true exudative pneumonia.

We must not, however, draw the line too absolutely between these two forms of pneumonia, for they are certainly not unfrequently mingled together; but it is the catarrhal pneumonia which is the important disease, the natural tendency of which is to break down into cavities, while any croupous pneumonia with which it may be complicated

¹⁷ The breath-sound which is audible over a portion of lung when the consolidation is not uniform, but in scattered nodules, is variable within certain limits; sometimes it is simply harsh, sometimes bronchial, but with some vesicular murmur super-added. I find the term "broncho-vesicular" a convenient one briefly to describe this sound. It also very well describes the sound frequently heard over a small cicatrising cavity with compensatory vesicular enlargement around it. When the nodules are large enough to be mapped out by percussion, of course the bronchial breath-sound will be correspondingly isolated.

readily undergoes resolution. We thus, from clinical and *post-mortem* experience, know that an acute apex pneumonia is of a much more serious kind than a basic one—it should be always regarded as probably a phthisical pneumonia, and although I cannot strictly speaking agree with those observers who say that the inflammation has been determined to that part by the presence of tubercles, yet, clinically and in their acceptation of the term tubercle—as including both the miliary granulation and nodules of catarrhal pneumonia—this view will I think generally hold good.

The main points of the case before us may be summed up as follows:—A woman with a tolerably strong hereditary tendency to consumption (her mother was probably phthisical when she was conceived), enjoys fair health until she is 27, but in the course of the succeeding two years and a half she gives birth to three children in two confinements, two of whom live and are suckled by her. Shortly before the birth of her last child her health breaks down altogether, and she becomes the subject of phthisis. It is to be remarked that there is no history in this case of any exposure to cold.

Now, I think it is fair to assume that, had this patient been in happier circumstances, had her health not been depressed by the development at her expense of three infants and the maintenance of two of them, while she herself was doubtless not in the enjoyment of nutritious food in any great abundance, had she sought advice earlier (and taken it), she would never have become phthisical—her hereditary proneness to consumption might have remained a mere latent tendency. Had there been any family tendency to insanity, it is quite possible that the same evil conditions which have now led her to become phthisical might have caused her to be afflicted with some form of puerperal mania. "It is true that privation, excess, errors in habits of life, the sedentary occupations, the pernicious influence of certain trades, grief, anxiety, and other wasters of vital power, will not suffice to induce consumption

in all, or even in the greater proportion of individuals; for these agents, so universally prevalent, are part of the daily lot or of the daily errors of many more than fall victims to consumption. But it is also true that if to any or all of these conditions that of inherited tendency to phthisis be super-added, very few indeed escape the disease."¹⁸ The truth of this remark is well borne out by the above, amid numberless other cases which must be familiar to physicians.

The points about the case which rendered the prognosis a favourable one, with certain reservations, were:—1. The obvious and very sufficient determining cause. 2. The limitation of the disease to one apex. 3. The presence of considerable crepitation and some dulness, without any local flattening or marked difference in expansion. 4. The absence of fever at the time of coming under observation.

1. If the circumstances of the patient admitted of complete rest from the cares and anxieties of her position of life, and a change to a purer air, there would scarcely be a doubt as to the prognosis; and although she has had no advantages of this kind, and with the common dread of her class of becoming again pregnant, she could not be persuaded completely to wean her child, she has yet greatly improved; the disease has not extended, the physical signs show drying up of secretion sounds and pulmonary collapse.¹⁹ The encroachment of the margin of the opposite lung, and the gradual appearance subsequently of some flattening, show that the lung beneath is cicatrising. This encroachment of the margin of the opposite lung towards the diseased side should always be anxiously looked for; it can be readily made out by percussion, and when the disease is one-sided, it precedes, often by a long interval, any decided apex flattening.

¹⁸ Dr Pollock, *Elements of Prognosis in Consumption*, p. 340.

¹⁹ Since the above was written this patient had a second distinct attack which resulted in further damage to the left lung and from which she again rallied, but died some months later.

3. The late appearance of flattening—coincidentally, that is to say, with the lessening and disappearance of moist sounds which have been considerable—is an important sign of arrest of the disease, in contradistinction to flattening which comes on coincidentally with an advance in the other physical signs, and which may be due, therefore, to sheer loss of lung substance, or to the presence of the indurative form of disease, chronic pulmonary tuberculisation, which, though of chronic course is yet one of the most intractable of lung affections. Flattening must then only be considered in conjunction with other signs, and with especial regard to the period of its appearance. In the case before us, it signifies together with the other signs, pulmonary collapse, with perhaps a few shrunken nodules, adhesion and some thickening of the pleura.

I believe that most cases of “cured” early-stage Phthisis are of the kind above related. The cure may remain permanent but one must always bear in mind that catarrhal pneumonia is one of those diseases that are peculiarly prone to recur, resembling acute rheumatism, tonsillitis and some other diseases in this respect.

The delicacy of the lungs inherited or acquired which has led to the first attack remains, nay, is increased by that attack. Hence the previous health history of the patient helps us much in the prognosis in each individual case.

Those cases in which the pulmonary delicacy is distinctly inherited are the least hopeful, those again in which the attack has been most distinctly led up to by adverse conditions of a definite and remedial kind are the most favourable. Of course in each case the extent of lung involved and the intensity of the disease, to be ascertained only by physical examination, must as already pointed out most importantly enter into the question as to prognosis.

As soon as the fever has passed we may accurately gauge the degree and amount of pulmonary loss and taking all the

circumstances into consideration a tolerably sound prognosis may be arrived at.

It is not uncommon to meet with cases in which the second attack does not affect the same part of the lung as the first but the opposite apex or some other portion of the same lung.

I have quite recently seen a young married lady who, six years ago consulted the late Dr. Salter for incipient "tubercular" disease affecting both apices and was recommended by that physician to go to Australia. She did so, her husband, a medical man, giving up his practice for that purpose. Her cough disappeared by the time she reached Melbourne and she has enjoyed excellent health there since, save in the hottest season of the year. There is now very distinct flattening below the left clavicle with somewhat harsh and feeble breath sound and slight dry crackle after a forced cough. She does not complain of cough, however, and appears in all respects well. The right lung is considerably expanded. I take it this was originally a case of alveolar catarrh affecting both apices and in the left side proceeding to catarrhal pneumonia of the second degree (p. 7) now obsolescent.

With regard to the management of such cases as these, there is little comment needed. It is unnecessary to point out the great importance of complete removal for a time, at least, from the adverse circumstances which in the first place induce the disease. In the case of women who are suckling, a partial weaning of the child is of very little use; the irritation and vascularity of the breasts is kept up by the occasional lactation, and the nutrition of the patient is diverted from its natural objects almost as completely as before.

The exact value of rest as obtained by such mechanical appliances as the "lung splints" first employed by Dr. Dobell,²⁰ or the method of strapping the chest adopted by Dr. McCrea²¹ of Belfast, needs, at all events as regards apex disease, to be further tested. The naturally very slight expan-

²⁰ *Brit. Med. Journ.*, Nov., 1873.

²¹ *Lancet*, 1874.

sile movements of the apices become still further restricted in disease without mechanical aid.

I have tried in a few cases at the Brompton Hospital the effect of restraining the movements of the chest in apex catarrhal-pneumonia by keeping the arm on the affected side in a sling. It is difficult to say that any distinct advantage has been derived from this treatment but it is undoubtedly advantageous in so far as it prevents any strain upon the lung that would be produced by an active employment of the arm as in reaching or lifting. During the first febrile stage of the disease it is of great importance to keep the patient in bed or at least on the sofa, for every, even the slightest, effort in such cases hastens respiration.

In inflammatory basic complications which are very apt to supervene in the course of phthisis, I have very frequently obtained good results by restraining the chest movements by the application of a broad piece of strapping of sufficient length to extend round the semi-circumference of the chest, and for an inch or two beyond the median line in front and behind. I have not found it necessary in such cases to adopt the more thorough method of strapping the chest, recommended by my colleague, Dr. Fred. Roberts,²² in the treatment of pleurisy. Immediate relief to pain and amelioration of the local signs usually follow this treatment. If necessary, local anodynes or counter-irritation may be combined with mechanical rest, by including underneath the strapping a belladonna plaster or a piece of spongio-piline sprinkled with dilute Iodine or some such application.

Mild preparations of Iron are usually needed in these cases and one of the best forms of iron to begin with is the ammonio-citrate, to which a little aromatic ammonia is added.

There is one symptom in the above-related case which deserves a special comment; the cough remained troublesome

while the pulmonary signs were greatly improving and all secretion sounds rapidly drying up. Dr. Thorowgood²³ has drawn attention to the irritable dry cough which is so frequently attendant upon the subsidence of pulmonary disease, and truly observes that the patients should be encouraged to check the cough themselves as much as possible. This they can do to a great extent, and may be assisted by some sedative cough mixture, if necessary, to secure rest at night. The morning cough in these cases—and, indeed, in many others—is the most troublesome. It is, however, the natural consequence of a good night's rest, and should never be checked by a sedative, since the retained matters suitable only for expectoration considerably impede respiration, become highly irritating, and much increase the future trouble from cough. A cup of warm cocoa, or tea, or milk, taken before rising, will greatly facilitate expectoration. If this does not suffice, a simple steam inhalation is useful, or a small dose of ether and ammonia.

²³ *The Climatic Treatment of Consumption.*

CHAPTER V.

Case of Catarrhal Pneumonia, non-hereditary—Significance of Fever and of the signs of softening; their relation to one another—Gradual change in Physical Signs; Supervention of Pulmonary Fibrosis—A second case briefly referred to—Further progress of such cases; a hint as to their management.

It will, I think, be most convenient to follow up the remarks upon the case described in the last chapter, by detailing one or two other cases which will serve to illustrate the transition, which not very unfrequently occurs, of pneumonic into fibroid phthisis. Bearing in mind the relation of the tissues to one another which are affected in these two forms of phthisical disease as pointed out in the second chapter, we perceive that the transition of the one form into the other is pathologically very easy; it is also, I am persuaded, often to be observed clinically.

I do not of course mean to contend that all cases of fibroid phthisis commence as catarrhal pneumonia; the question as to the ætiology of fibroid phthisis is not for the moment before us. I only wish to refer to catarrhal pneumonia as *one* of the several processes upon which fibrosis of the lung may supervene.

John B., aged 29, a butcher's assistant, came under my notice in March, 1871. He was a broad-chested, powerfully-made man, of medium height and florid complexion. He had led a rough but sober life, having followed his present business, which included the slaughtering of animals, for some years in Australia, and had enjoyed excellent health until shortly before Christmas, when, after getting wet, he caught a severe cold, which was followed by a cough, which had since increased, unaffected by treatment. Up to and at the time of his attendance, however, he was still following his

employment, but he now did so with difficulty, complaining of his cough and of increasing weakness with decided emaciation. His father had died of consumption, brought on, the patient stated by intemperance; there was no other hereditary tendency to the disease.

The chest, as before said, was broad and well-formed, without flattening or obvious impairment of expansion. The heart's apex beat in the natural situation. At the left clavicular and subclavicular region the percussion note was dull, the dulness extending to the fourth rib; posteriorly, the resonance was defective at the left supra-spinous fossa. Scattered over the dull regions there was coarse crepitation, mingled with a still larger humid crackle. These moist sounds were abundant, and masked to a great extent the respiratory murmur, which was decidedly harsh, but not distinctly bronchial. Its vesicular quality became gradually restored as the stethoscope was passed downwards. At the posterior base there were some scattered sibilant râles. On the right side the percussion note was good, and the breath sounds were natural.

The disease in this case began then, with a cold on the chest—*i.e.*, a more or less general bronchial catarrh—which became localized²⁴ at the left apex, and extended there into the alveoli, producing catarrhal pneumonia; yet he continued his daily work, though constantly losing strength, for three or four months, during which time the catarrhal process ran on insidiously to a more deeply inflammatory degeneration of the alveolar walls. The disease had been acquired by exposure, the family tendency being very slight.²⁵ The whole

²⁴ This bare statement is a clinical one only. We find in almost all cases of phthisis one apex affected first. Why cachectic bronchial catarrh should so strongly differ in this respect from ordinary bronchitis remains a mystery, but the fact is not the less clinically important.

²⁵ The father of this patient acquired the disease from his intemperate habits since the patient's birth.

build of the chest was not that of a man who inherited any tendency to phthisis. The physical signs at the present stage showed consolidated lobules of blocked alveoli, which were softening with varying degrees of rapidity ; the coarse crepitation answering to the redux crepitation of pneumonia, the larger click being due to more profound destruction of tissue (softening).

There were two data defective in this history :—1. Fever. 2. Rusty sputa, or haemoptysis. Although, however, the patient could give no definite and trustworthy information as to fever, we know for certain that he must have had intermittent attacks of fever ; for it is a matter of clinical experience that we never get the signs of pulmonary consolidation and resolution or softening, dulness and crepitation, without there having been with each increment of pneumonia a period of elevation of temperature. A remarkable case well illustrating this occurred some two years ago in the Brompton Hospital under Dr. Sanderson's care, in which a woman had for weeks together daily attacks of fever of an intermittent character, and *post-mortem*, one of the lungs was completely, and the other partially, consolidated, and as it were mapped out on section by lobular pneumonia of different dates. The patient whose case we are now considering had, on the occasion of his first visit, a quick pulse and a somewhat red tongue, and though there was no elevation of temperature at the time, it is very likely that at night it was a little raised

It is here, however, perhaps, well to remember that we may have dulness and large or small liquid rhonchus without there being any fever present *at the time* ; the fever may have passed away, but the consolidation which accompanied the fever cannot disappear so rapidly, but must run through a series of pathological and chemical changes essential for its removal by absorption or elimination—changes which are not accompanied by fever, and which may proceed *pari passu* with the rebuilding of the frame exhausted by the previous fever.

This reflection is of very great importance in a therapeutic point of view; for if, for instance, we were to treat ordinary basic pneumonia with anti-pyretics so long as the stethoscope revealed tubular breathing and moist sounds to be present, we should get results deplorable in direct proportion to our auscultatory skill; and this remark applies with equal force to the lobular consolidations and softenings of phthisis. We should be acting as foolishly, if we regarded them as indications for treatment of an antiphlogistic kind, as if we continued to wrap up our damaged water-pipes after the thaw had set in instead of hastening to adopt measures to repair the breach as soon as the frost had gone.

Rusty expectoration is by no means a constant symptom of catarrhal pneumonia—not so constant, indeed, as it is of the croupous variety. It appears to depend partly on the degree of intensity of the pneumonia and the congestion with which it is attended, but also very largely upon the constitutional peculiarities of the patient.

To pursue the case, however, one step further. The patient was treated with an alkaline mixture containing small doses of iodide of potassium, and with cod-liver oil. The next note of importance was taken on April 27, when the expansion of the left side of the chest was noted to be decidedly impaired, the dulness had increased in hardness but not in extent, and was very marked, especially between the left margin of the sternum and the mid-clavicular line.

In the space marked out by these two vertical lines, (left sternal and mid-clavicular), the respiration was extremely feeble, and not attended with any rhonchus; the heart's impulse was diffused to the second inter-space, though the apex was only half an inch higher than natural. To the left, again, of the mid-clavicular line the respiration was still feeble, and the rhonchus much diminished, the dulness being somewhat increased. At the apex posteriorly there was bronchial respiration and imperfect pectoriloquy; the bronchial râles at

the base had cleared up. The resonance of the right lung extended to the left margin of the sternum.

These signs showed — 1. That the disease had not extended; on the contrary, the signs of bronchial irritation at the base had cleared up.

2. A wasting of the parenchymatous texture of the lung had taken place; degeneration, absorption, and expectoration had removed the morbid contents of the alveoli, and some of the lung tissue itself, leaving, perhaps, at the apex a small cavity; the general result being collapse and agglutination of air-cells. Hence a considerable reduction in the bulk of the lung and the retraction of its anterior margin *away from the median line*; so that between the left sternal line and a line drawn from the point of junction of the inner and middle third of the clavicle to the apex of the heart there was probably at this date no lung at all.

3. An encroachment of the enlarging right lung, a slight shifting of the heart to the left, and a flattening of the chest wall to make up for the lost space. The chest wall flattening was, however, very slight, and not yet noticeable until the patient drew a breath. The man had powerful parietes, and in such cases the displacement of heart and encroachment of the opposite lung precede, often for a long time, any obvious flattening.

It was remarkable with what rapidity these changes were taking place, and there can be no doubt that the connective tissue of the bronchial and perivascular and pleural sheaths were undergoing rapid development, and that the case was at this date not merely one of catarrhal pneumonia which had subsided after having caused a certain loss of lung substance but that an interstitial pneumonia was proceeding; the case had changed its type to one of peribronchial, or rather pulmonary, fibrosis; it had lapsed into one variety of fibroid phthisis. That the disease was not yet arrested seemed probable from the patient still losing slightly in weight and becoming more anaemic; but it had clearly become limited.

During the next month he lost two pounds. He was during this time taking an acid preparation of iron, with a little quinine and the oil. Notwithstanding this slight loss of weight, he had improved generally; cough and expectoration had diminished, and he felt stronger. On June 8 he was still better, and had gained one pound since last report. He had very little cough; all moist sounds had disappeared except a slight friction (?) on cough at the outer side and a little above the left nipple. He has since steadily improved. (August 1871).

Dr. Andrew Clark related at the Medical Society a few months ago (*vide Lancet*, May. 6, 1871) a case very similar to the above. The subject of his paper, a boy of 14, had been a patient of mine at Brompton some five or six months before he came under the notice of Dr. Clark, and I took the same view as to the condition present, which Dr. Clark described by the name "peribronchial fibrosis." The case appeared to me, however, in its earlier stages, to be one of catarrhal pneumonia, undergoing the process of softening, upon which pulmonary induration supervened, in consequence partly of ordinary cicatricial collapse, partly from the vascular bronchial and pleural sheaths taking on an active growth. I should prefer to regard it therefore with that above described, as belonging to a section of the large class of pulmonary fibrosis, or fibroid phthisis, since, though in both cases there was undoubtedly at first more or less general bronchial catarrh, still it was the apex pneumonia, of an intensity sufficient to deeply affect the fibrous framework of the lung at that part, but not sufficient to lead to its destruction, which set going, as a subsequent result, that active hyper-growth which produced such important modifications in the morbid structure and physical signs. As I look upon this new disease, or phase of the disease, as affecting the whole framework of the portion of lung involved, I think the more general term pulmonary fibrosis a better one under which to include it.

This disease—fibrosis—has, I think, little or no tendency to spread beyond the limits of the original lesion which gave rise to it, and provided the patients are careful to keep up their general health, and to avoid fresh catarrhs, they do well. The indurated portions of lung usually enclose some nodules of dry cheesy matter, and one or two small excavations, which are often the seat of a necrotic crumbling process, which continues for a long time without affecting the general health, merely causing a slight irritative morning cough.

This crumbling process is, however, very favourable to the uncovering of vessels of considerable size, without their becoming obliterated by the coagulation of their contents. Small ectasias of these pulmonary branches, or even considerable aneurisms, are thus more apt to arise in these indurated lungs than in others, and the possibility of severe or fatal haemoptysis must not be absent from our minds in framing a prognosis.²⁶ Also, haemoptysis occurring in cases of this kind must for the same reason be regarded with greater anxiety than in ordinary cases of phthisis.

There is only one special remark concerning the treatment of these chronic indurative cases of phthisis during the often extended period of quiescence, which seems called for, and it is this—that though such cases require careful *surveillance*, and for several years, where practicable, carefully selected climates to suit the different seasons of the year, they do not require the persistent administration of tonic medicines and cod-liver oil. They improve immensely under such remedies up to a certain point, which may be readily recognised by the medical attendant, and cannot be better described than by saying that it amounts to the most perfect health attainable by a patient who has had a certain area of respiratory surface cut off. If beyond this point we persevere with iron and oil and too nourishing or stimulating a diet, we may still further

²⁶ I have treated this subject somewhat fully in the last volume of the *Pathological Transactions*, pp. 41-65.

increase weight and heighten colour, but the pulse quickens, the patient gets more short of breath; he becomes, in a word, plethoric, and liable to pulmonary congestion and hæmoptysis, or to dyspepsia and diarrhœa; and a rapid neutralisation of all the good results obtained, with great danger of fresh and perhaps fatal renewal of the old disease, is the consequence of too great anxiety, both on the part of the patient and the doctor, to again arrive at a degree of health and bodily vigour which is impossible with a permanently damaged lung. I have repeatedly noticed the arrival of patients at this stage of constitutional balance, and have witnessed the quickened pulse and returning fever consequent upon the indiscreet endeavour yet further to urge on the health with tonics.

Having thus touched upon the subject of Fibroid Phthisis and shown how that contractile variety of the disease may arise secondarily by gradual transition from the ordinary catarrhal pneumonic variety, I will complete what I have to say about this form of phthisis in the next chapter, in the course of some comments upon a typical example illustrating its main features.

CHAPTER VI.

Fibroid Phthisis, its characteristic signs and pathology summarised; reasons for selection of term as most applicable—A disease secondary to some parenchymatous affection of the lung, doubtful if pleurisy or bronchitis alone will give rise to it—Relations to other forms of phthisis—Example related—Summary—Remarks on diagnosis, prognosis, and treatment.

CERTAIN forms of pulmonary phthisis of various origins are attended by such an amount of interstitial fibrous growth as to give them clinical features of a very peculiar type.

The prominent symptoms and signs by which such cases are distinguished,—increasing contraction and immobility of the side, dragging pains, traction of organs to that side, deadened percussion-note and weakened respiration of more or less bronchial quality, intensely so, or cavernous, at parts; breathlessness, paroxysmal cough, occasional hectic, but general absence of fever, very chronic progress, long continued one-sidedness of the disease, and correspondingly slow failure of nutrition—show them to come within the definition of phthisis, but phthisis of a special kind.

The conditions presented to us, *post-mortem*, of a contracted, toughened, indurated, and usually pigmented lung, surrounded by a greatly thickened adherent pleura, containing one or more rigid, dense-walled cavities, dilated bronchi, and cheesy encapsulated nodules, are confirmatory of this view.

On minute examination, we further discover this condition of lung to have been produced by a growth of two kinds pervading it. 1. Connective tissue proliferation, resulting in the formation of bands and processes of *fibrous* tissue, derived from the sheaths of vessels and bronchi, and the sub-pleural and inter-lobular tissue of the lung. 2. A more important nuclear growth leading to the formation of broad tracts of *fibroid* tissue, thickening the walls of the alveoli, compressing,

and finally effacing them, unless they shall have been previously stuffed with their own catarrhal products; this fibroid structure being very possibly derived from the lymphatic elements normally pervading the lung.

The products of these two processes become intimately mingled, but it is the latter which is the phthisical element in the disease, for mere connective tissue growth does not lead to organic destruction of the lung. This is also the element which specially gives to the disease its peculiar clinical features, and renders the name "fibroid phthisis" applicable to it.

The term "fibroid phthisis" has been productive of much discussion. Originally introduced by Dr. Andrew Clark as "embracing all those cases, whether local or constitutional, which are anatomically characterised by the presence, in a contracted and indurated lung traversed by more or less dilated bronchi, of fibroid tissue, and of tough fibrogenous substance, together with cheesy deposits or consolidations, and usually small cavities, commonly found about the middle and lower parts of the affected organ"²⁷—it has been objected to as signifying too definitely the existence of the disease as a substantive or idiopathic one. Dr. Clark is, however, convinced that it may, and often does, appear as a primary affection, in which view he differs from most contemporary authorities. Still, the term is such a neat, concise, and clinically useful one, that it has been very generally accepted with the above reservation.

Dr. Clark argues, with much force, that these cases are indisputably cases of phthisis, while all their special characters are due to the pervasion of the affected lung by a contractile fibroid and *fibrogenous* tissue. These two facts cannot, I think, be controverted; and when it is further urged, as I think it may truthfully be, that this fibroid tissue is indistinguishable in situation and development from chronic tubercle, a very fair case may be made out for the preservation of the

²⁷ Clinical Transactions, vol. i. p. 188.

term "fibroid phthisis" though its distinguished author would, I fear, not admit this latter argument.

I ventured, in a paper read before the Clinical Society in October, 1868 (*vide Clinical Transactions*, vol. ii., p. 193), to question whether this term was advisable, since the pure constitutional disease was of such rare and doubtful occurrence. Later reflection and the observation of a good many cases have, however, led me to think that this objection, though by no means weakened,²⁸ is not sufficient to render the term clinically inapplicable, since "fibroid" only corresponds with other adjective terms—"catarrhal," "tubercular," etc., in describing the *predominant* character of the variety of phthisis thus distinguished; for in all cases of chronic phthisis the morbid processes are of more or less mixed character.

Dr. Wilson Fox in the exhaustive article above referred to, includes under the term "chronic pneumonia" all cases of pulmonary fibrosis uncomplicated by tubercle: his chronic pneumonia would therefore agree with Dr. Clark's fibroid phthisis, except that he does not admit the disease as a primary or constitutional one. On the contrary, he contends that it is always associated with, and dependent upon, catarrhal pneumonia; hence his employment of the term "chronic pneumonia." Dr. Fox though thus characterising the typical disease as essentially one of pneumonia affecting the lobules of the lung, not the connective tissue which binds them together, acknowledges that freedom from tubercle is very exceptional.

Dr. Bastian on the other hand, would include all these diseases under the old term "cirrhosis of the lung." Cases to which the term "cirrhosis" is strictly applicable, in which there is no catarrhal pneumonia or tubercle, nor any true organic destruction of the lung are, however, so rare as to render this latter name a misleading one.

²⁸ See article by Dr. Wilson Fox on Chronic Pneumonia in Reynolds' *System of Medicine*, vol. iii. p. 772.

This disease, then—fibroid phthisis—is in the great majority of instances, so far as my own experience informs me, of a truly secondary nature, supervening upon some more or less acute inflammatory affection of the lung, whether simple basic-, or broncho-, or catarrhal, or tubercular pneumonia. It is very doubtful whether pleurisy or bronchitis alone can give rise to it without the intervention of lobular pneumonia or tubercle. Local injury or pulmonary abscess may form the starting point of the disease, but as before observed are not alone sufficient to cause any extensive fibroid invasion of the lung beyond their own immediate limits.

Numerous examples may be found of this somewhat inclusive disease, ranging from the most typical cases to those which are indistinguishable from ordinary chronic phthisis. In the last chapter I described a case of catarrhal-pneumonic phthisis, in which the transition into one of fibroid phthisis or pulmonary fibrosis was traced. It would not be difficult to find examples in which the reverse takes place, the clinical characters of fibroid phthisis being gradually changed by subsequent pneumonic processes, and all the features of the special variety becoming merged in the diffuse pulmonary destruction.²⁹

Cases of fibroid phthisis may be roughly divided for convenience of clinical exposition into three varieties :—1. Those in which the disease has its starting-point, at the apex of the lung, and is proceeding downwards. 2. Those in which it commences at the base, and advances upwards. 3. Those in which the most marked signs are discoverable about the middle of the lung. Of the first variety the following case is a fair example :—

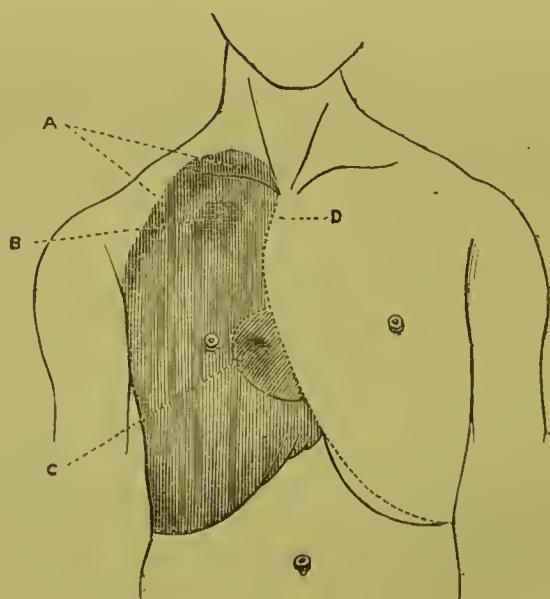
George P., a sawyer, aged 43, came first under my notice

²⁹ In the case of the boy referred to at p. 42 as having been described as one of peribronchial fibrosis, active destructive changes subsequently set in quite obscuring the original characters of the disease and the boy finally died with pneumothorax.

as an out-patient at the Brompton Hospital in August 1868. He was a thin man, with dark hair, having no hereditary predisposition to lung disease, except that his father had suffered from "asthma." In the preceding January, having suffered from slight cough for years, he was laid up for six weeks with "inflammation of the right lung." Since that time the cough had been continuous, and three months ago had been attended with slight haemoptysis. The cough was now paroxysmal, causing retching and often rejection of food; expectoration difficult, abundant, and of a pink tinge. He had got thinner lately. The digestive functions were fairly good; the pulse a little hurried; there was no fever present.

On inspecting the chest, cardiac pulsation was visible at the fourth right interspace, c, to left of right nipple. This side was diminished in size and much restricted in movement, the intercostal spaces deepening with inspiration; while the left side expanded freely, with an uplifting movement of the shoulder. On careful examination, the apex of the heart was found a little to the left of the ensiform cartilage.

On percussion, the right side in nipple-line anteriorly was dull to the second rib, comparatively resonant to the fourth,



and below this point it was again toneless. To the left of the line of sterno-clavicular articulation, at the level of the second and third cartilages, there was good resonance continuous across median line with that of the opposite side. The line of this resonance, D, sloped upwards to the episternal notch, in a downward direction being displaced by cardiac dulness at the fourth cartilage. Hepatic dulness barely reached the costal margin. There was dulness throughout the *axillary* region and *posteriorly* from apex to mid-scapula, the note having a tubular quality in this latter region. Below mid-scapular there was fair resonance to the ninth rib, though less and harder than on the opposite side; the lower two or three inches on the right side gave a flat note on percussion.

The percussion-note over the whole left side, including the region of normal cardiac dulness and extending across the median line, as above indicated, was full and good in front and behind.

The auscultatory signs were in agreement with those of percussion. Above the clavicle on the right side, the respiration was amphoric, and dry; below the clavicle weak and bronchial to the base, with some rather large, moist rhonchus, friction, and bronchophony. At one spot corresponding with the second and third ribs, nipple-line, B, the breath sound was of tracheal quality, with scanty cavernous clicks and pectoriloquy. In the upper axillary region the respiration was amphoric, and the voice-sound pectoriloquous; in the supra-spinous fossa and interscapular regions, cavernous-blowing, with pectoriloquy. Blowing respiration extended to the angle of the scapula, where it became weaker and gradually annulled at the base. The vocal fremitus was generally increased on the right side. Respiration throughout left lung exaggerated vesicular.

February 27th, 1869.—Patient improved in flesh and appearance; stronger than before, but complains much of cough, and expectorates much pink phlegm. Breath short on

exertion; cough causes retching, but does not bring up food now; appetite fair; digestion not very strong; bowels regular. Continues medicine (oil with nitro-muriatic acid and cinchona). Fingers noted (February 13th) to be clubbed.

The additional physical signs noted at this date were, a distinct short systolic bruit at the point of maximum cardiac impulse, not appreciably increased by pressure nor confined to that spot, being also audible at the apex. Measurements of chest :—

From mid-sternum to nipple, right side, 4 inches; semi-circumference, $15\frac{1}{4}$ inches; expansion, $\frac{1}{4}$ inch.

Left sterno-nipple measurement, $4\frac{1}{4}$ inches, semi-circumference 16 inches; expansion, $\frac{1}{2}$ inch.

We may, by way of summary, aided by a glance at the figure (reduced as accurately as possible from a sketch taken at the time upon an outline diagram), interpret the above detailed physical signs as indicating at this period a general induration of the right lung, with much contraction, its anterior margin having receded considerably from the median line, exposing the pericardium, and having also shrunk away from the upper surface of the liver. Its upper and a portion of its lower lobes were extensively excavated, the cavities being old, tolerably dry, and shrunken with the general contraction of the lung. The pleura, (from the hardness of percussion, feebleness of breath-sound, and great fixity of walls) was probably greatly thickened. The liver was drawn up within the costal margin, and the heart considerably displaced to the right, its axis being, however, but little altered. A short systolic murmur was heard over the heart.

Subsequently to the last note I repeatedly examined his chest during the many months of his attendance at the Hospital but beyond some variation in the dryness of the sounds there was no important change in the physical signs. I think I examined the urine more than once, but the only note I have of it was taken in April, 1869, when it was acid, became

slightly turbid on boiling, but cleared on adding a drop of nitric acid.

The left lung remained healthy, and though the patient continued thin and cachectic-looking with a troublesome cough, he held his ground fairly well, and rather improved in general health. At times the expectoration would become very abundant, and occasionally of a pink colour, (I thought due to fresh irritation and slight sanguineous discharge from the walls of the old cavity). The most troublesome symptom throughout the case—and one which is common in greater or less degree to all those cases of phthisis in which indurated thick-walled cavities are present—was the paroxysmal cough terminating in vomiting, occurring especially after meals.

No doubt the mechanical conditions of such a cavity, rendering the removal of expectoration very difficult, have much to do with the production of vomiting, and render it a particularly common symptom in these cases; but the reception of food into the stomach has seemed to me to be in many cases so constantly followed by cough ending in vomiting, as to render this mechanical explanation insufficient, and, in 1869, I was led to attribute it to an undue reflex irritability of the pneumogastric nerve, and proposed strichnia as the best remedy.³⁰ I have since, in many cases, found this remedy valuable, but by no means invariably so.

On leaving off attendance at the Hospital in May, 1869, the patient, though not free from cough, continued for a time to improve, but he soon afterwards began again to emaciate, and the vomiting with cough returned. He again attended in January, 1870, resumed the oil and used carbolic acid inhalations, and left March 30th, improved. Since this time I have heard nothing of him.

The above-related case represents very well the main

³⁰ *Practitioner*, vol. i., p. 312 :—Dr. Hughes Bennett (Reynolds' "System," vol. iii., "Phthisis," 1871) regards this as a cause of vomiting in the later stages of phthisis.

features of fibroid disease of the lung. The indurative disease supervened presumably upon an acute apex (tubercular or catarrhal) pneumonia, and did so with tolerable rapidity, the characteristic symptoms and signs being fully developed within six months of the termination of the acute disease.

The question as to the rapidity with which this disease may advance is one of great interest, and requiring further observation. I cannot but think that, reasoning from the morbid appearances found *post-mortem*, we are apt to regard such diseases as older than the clinical history will warrant us in believing; on the other hand, though it is very probable that the fibroid induration of the lung may proceed with great rapidity to such a stage of shrinking as to produce very marked clinical signs, its subsequent progress is very slow and difficult to measure, consisting mainly in the further hardening of an already indurated lung, the gradual widening of the bronchial tubes, and filling up of the loose oedematous areolar tissue between the separated pleural layers by dense fibrous growth. We can readily perceive therefore how the earlier stages of the disease which are attended with striking alterations in physical signs may, if the original disease is of considerable extent, be passed through with comparative rapidity, while the later progress is necessarily slow and difficult to estimate.

The resonance of the opposite lung extending across the median line would emphatically exclude cancer, which the history of the case, and many other signs, particularly the kind of cardiac displacement, would also negative. While in certain cases of mediastinal cancer the heart is fixed in about its normal position, it is, I believe, never displaced towards the side most affected. In regarding the signs of cardiac displacement, however, it is important to avoid taking the point of maximum impulse as necessarily the apex beat. In this case the real displacement of the heart is much less than a first glance would lead us to believe. The absence,

while the patient was under observation, of any evidence of complication of other organs would be in favour of the disease being of local origin.

In calculating the *prognosis* in these cases, we have to bear several things in mind—the cachexia of the patient, the size and freedom of communication with external air of any cavities present, and the evidence of disease of other organs, especially of the opposite lung.

The cachexia is occasionally very manifest; without very marked emaciation the anaemia is apt to become great, and the complexion of a straw-tint, reminding one of that seen in the later stages of certain cases of cancer, or in women suffering from uterine disease. None of these peculiarities were in the present case noticeable in any marked degree.

A cavity of considerable size, and freely communicating with the outer air, is a more hazardous condition for the patient than one which is small, or which we may presume to have become flattened and partially or completely closed. In the former condition the patient is constantly liable, on exposure to cold etc., to recurrence of irritation and fresh ulceration of the cavity-wall causing profuse secretion and hectic, as in the present case, or laying bare vessels which may at any time rupture and cause death from haemoptysis. Out of eight well-marked cases of which I have made *post-mortem* examinations, this has been the cause of death in two.³¹

When the opposite lung is affected, it is most generally by grey tubercle, and it becomes so affected sooner or later in most cases, unless the patient be cut off too soon by some intercurrent disease. We must, however, not give a too hastily fatal prognosis from mere physical signs in such cases, for the course of the tubercle is disposed to be very chronic and indurative, and the signs may again subside and long remain

³¹ For an account of one of these, Downer, vide *Clinical Transactions*, vol. ii., p. 181; the other is referred to in a Table on Haemoptysis, *Pathological Transactions*, vol. xxii., p. 58, Case 1, F. W.

in abeyance. I still occasionally see a patient whose case (Case II) I brought before the Clinical Society in 1868. He had then distinct evidence of involvement of the apex of the opposite right lung and also a small quantity of albumen in the urine. He has since, however, led a rural life and his health has yearly improved. The right lung has become greatly enlarged, the disease in the left remaining perfectly obsolescent and the patient is now (1876) in good flesh, and, to all appearance, in robust health. But when involvement of the opposite lung is attended with decided elevation of temperature, the prognosis must be most guarded, for the supervention of pulmonary tuberculosis is perhaps the most common cause of the fatal termination in these cases.

Dr. Clark regards albuminoid degeneration of other organs—the liver and kidneys—as commonly supervening in the later stages of this disease. Of the three purest cases of which I have made *post-mortem* examinations, in which there was no obvious grey tubercle in the other lung (though islets of peribronchial induration were present in all), in one there was extensive albuminoid degeneration of liver and spleen with granular kidneys; in another of the spleen only, with ulcerated intestines. Albumen in the urine, absent in the present case, affords us the earliest clinical evidence of this degeneration.

Although the prognosis is always, in cases of fibroid phthisis, a precarious one, from the circumstances above mentioned, yet the course of the disease may be very long, and may with due precautions in some instances be almost indefinitely extended. The condition of health and *physique* maintained by some patients is remarkably good. In this respect the example above related is an unfortunate one; I have notes, however, of a postman presenting exceedingly well-marked signs of this form of phthisis, who almost entirely lost his cough while attending the Hospital, and was able to resume his duties, walking fourteen or fifteen miles a day; and I

might mention two or three other patients capable of considerable physical exertion on level ground. Patients who for many years have had one lung dormant or "gone," as they usually describe it, are not uncommonly met with and belong to this category. I have already mentioned one such case which I described at the clinical society some years ago, and I occasionally see a lady who has for twenty-five years had her left lung similarly affected. This patient leads a sheltered but useful life and rarely suffers from pulmonary symptoms of any kind: her chief complaint, from time to time, being of failure of heart's action causing chilliness and a disposition to fainting with occasional attacks of great cardiac oppression which, but for the absence of any severe pain, would be described as angina. There is no evidence in this case of valvular disease or decided dilatation of heart but the organ is uncovered by the retracted left lung and the sounds are feeble. I have observed in many chronic left sided cases of phthisis great functional disturbance or rather irritability of heart doubtless attributable to its being less supported and protected by lung than in health.

The management of this variety of phthisis calls for no special remark; the prevention of fresh catarrhs by judicious clothing, the selection of climate when practicable, the avoidance of night-air, and protection from irritating fogs or cold or damp winds by respirators, are, with a nutritious but not stimulating diet, the hygienic measures to be adopted. Iodine frictions, soothing or antiseptic inhalations (carbolic acid being particularly useful when there is any foetor of expectoration), seem the best local remedies; whilst the general condition, including that of digestion, the nature of the cough and amount of expectoration, supply us with indications for the administration of appropriate drugs—iron, cod-oil, strychnia, alkalies, tonics, etc.,—or warrant the withdrawal of all medicines.

The very full consideration of the above example of fibroid phthisis, and of the general bearings of the disease, renders

it perhaps unnecessary for me, to extend this already very long chapter by entering into those variations in the signs of this form of phthisis which depend upon its seat of origin. I will only observe that I believe the commencement of the disease in the central portion of the lung to be of very rare occurrence, much more rare than either at the apex or base. It is true that the most advanced physical signs may often especially in left sided cases be found at the 3rd or 4th ribs, outside the nipple line and in the upper axilla, but this localisation of signs is acquired by the lowering of the apex, and shrinking away of the lung from the median line in the course of the contractile disease.

There are a few points for consideration in the *diagnosis* of the above case.

That it is not simply a case of contracted cavity is evident from the contraction of the side being general, from the heart being displaced laterally, not specially drawn up towards the right apex, and from the weakness of breath-sound, with dulness at the base, and raising of the liver. The presence of considerable excavation at the apex would be in favour of the disease having commenced there as an apex (phthisical) pneumonia, and not being secondary to basic pleuro-pneumonia or empyema. The same circumstance would also distinguish the case from one of simple cirrhosis of the lung.

CHAPTER VI.

On Hæmorrhagic Phthisis—Subject divided into Hæmorrhagic Phthisis proper and Recurrent Hæmoptysis : distinction between the two—True hæmorrhagic phthisis very rare ; difficult to ascertain the true relation of the hæmoptysis to the primary disease : views of Niemeyer—Illustrative case of hæmorrhagic phthisis—Remarks : (1) Why considered a case of phthisis ; (2) Why hæmorrhage not bronchial, and affecting lung secondarily ; (3) Why probably coincident with and caused by active pulmonary congestion ; general conclusion, with definition of Hæmorrhagic Phthisis ; further points for and against hæmorrhage in such cases being bronchial—Disease has no relation to Hæmorrhagic Diathesis—Remarks on Treatment ; value of thermometer as a guide.

The term “hæmorrhagic phthisis”³² has crept into use to distinguish those cases of phthisis which have a distinctly hæmorrhagic origin, and also those in which, however they may have arisen, hæmoptysis is a marked and oft-recurring symptom. For clearness sake it will, I think, be better to restrict the term to the first set of cases to which it was originally applied—those of hæmorrhagic origin ; and to refer to the second class later as cases of phthisis with recurrent hæmoptysis.

The question as to the *frequency* with which phthisis arises as the direct result of hæmoptysis is one of great difficulty to

³² “Phthisis after Bronchial Hæmorrhage” (Niemeyer, Burger, 1864) “Phthisis Hemoptoique” (Herard and Cornil, 1867), “Hæmorrhagic Phthisis” (Dr. Williams, 1868). “Tuberculose nach Hæmoptoe” (Waldenburg, 1869), “Hæmoptysical Variety” (Dr. Peacock, 1870). These several authors, though doubtless they would agree in acknowledging certain typical cases as representative of this hæmorrhagic form of consumption, would differ widely in the liberality with which they would group more doubtful cases among such representative ones, nor would they more closely agree as to the pathology of the affection. The observation of this difference of opinion among enquirers so eminent, is alone, I hope, sufficient to make me fully sensible of the difficulties of the subject.

determine, and must indeed, I think, ever remain one of opinion. Of the possibility of blood having obtained entry to the air-cells and coagulated there giving rise to broncho-pneumonia, and subsequently to phthisis, the observations of Professor Niemeyer, and Drs. Hermann Weber, Baümler, and others have furnished us with clear evidence. I have myself seen on several occasions *post-mortem* examples of inhaled blood forming the nucleus of fresh lobular pneumonia in the grey stage in cases—it is true, of tolerably advanced phthisis—in which death ensued after recent hæmoptysis.

The difficulty, of course, lies in determining whether the hæmorrhage is the cause of the disease or is itself the result and evidence of pre-existing or coincident disease. It is an undisputed fact that in a certain number of cases, more or less copious hæmoptysis is the very first symptom of the pulmonary disease, preceding, even for a considerable time, all reliable physical signs.

Professor Niemeyer asserts that the hæmorrhage in these, as in the majority of cases of phthisis in all its stages proceeds from the bronchial mucous membrane; that a portion of the blood becomes inhaled into the air-cells of the previously healthy lung, coagulates there giving rise to irritative lobular pneumonia, the consolidations of which may subsequently decay and soften leading to destruction of the lung, or may become cheesy, and give rise at some subsequent period to tuberculosis of the lung of a secondary or infective kind.³³ I must confess that I have never met with a case which I could distinctly refer to this category, nor are the cases which Niemeyer quotes in support of his view to my mind conclusive.³⁴

³³ Waldenburg, fully admitting this view, seems inclined further to think that the inhaled blood, by the re-entry of its shrivelled elements into the circulation, may give rise directly to true tuberculosis. *Die Tuberkulose*, p. 496.

³⁴ This point is very fully discussed by my colleague, Dr. C. T. Williams,

I think the phenomena exemplified in the following case may be fairly regarded as illustrative of the hæmorrhagic variety of consumption, while they also point to the hæmorrhage being of pulmonary, not of bronchial origin.

J. P., aged 20, a servant residing at Faringdon, came to see me at the Brompton Hospital on September 21st, 1871. She was a fine, well developed woman, with a decided tendency to *embonpoint*, a clear complexion, a high colour, in which, however, a slight degree of lividity was noticeable. She was complaining of shortness of breath and bad cough with expectoration, and had the following history:—Of healthy parentage, and with no hereditary tendency to consumption,³⁵ she had enjoyed good health—with the exception of the formation of an abscess at the top of the sternum, which discharged eight years previously, leaving a depressed scar—until twelve months before admission, when she began to suffer from palpitation on exertion; she did not consider, however, that she had been ill for more than six months, with increasing breathlessness and palpitation and slight dry cough.

Three months ago she had an attack of tolerably copious hæmoptysis which lasted nine days, and kept her in bed three weeks: five weeks ago she had a repetition of the hæmoptysis to a less degree. She had not menstruated for four months, having previously done so with regularity; but although the catamenia ceased about a month previous to the first attack of hæmoptysis, she did not herself connect the two facts together; they had again appeared a day or two ago. She had never suffered from epistaxis. She now complained of shortness of breath, troublesome cough with expectoration, which, however, was not tinged with blood. She had got

in his chapter on Hæmoptysis in the recent work on *Pulmonary Consumption*, by Dr. C. J. B. Williams and himself. Five years further experience only enables me with greater emphasis to reaffirm the above statement.

³⁵ There is some doubt about one sister who died of bronchitis, after about six weeks' illness, at the age of 4 years.

thinner of late, and the pulse was rather accelerated and small.

There was at this date no alteration in the shape of the chest, which was remarkably good, nor any decided dulness; but on the right side there was diffused crepitation throughout, mingled with vesicular harsh breath-sound. On the left side the respiratory murmur was exaggerated. The case was regarded as one of irritative catarrhal pneumonia of the right lung, secondary to copious hæmorrhage (probably vicarious) from the apex of that lung. At this time there was no evidence of positive destruction of lung, and I strongly advised her to seek the shelter of the Hospital as an in-patient with the hope that she might make a complete recovery. She was not able to do so, however, until November after having had a slight return of the hæmoptysis at the end of October.

November 17.—Having been in the Hospital for a few days, under the care of Dr. C. T. Williams, I again examined her. There was slight but decided dulness on percussion at the right supra- and infra-clavicular region, fading both downwards and laterally towards the median line into good resonance; above the clavicle the resistance to percussion was greater than on the opposite side; at the extreme posterior base there was almost complete dulness for three fingers' breadth, not so anteriorly nor in the axillary region. Heart's apex-b at in normal situation. The respiratory murmur at the apex was entirely masked by coarse moist crepitation accompanying both inspiration and expiration; vocal resonance increased, however, in supra-spinous fossa. Below the apex respiratory murmur feeble; crepitation to base, but less abundant than above; at posterior base absence of respiration over the dull portion, with some incomplete ægophony. On the left side respiration loudly puerile, well audible to mid-sternal line. The complexion was still fresh and highly coloured, without, however, any lividity. Cough now only slight; expectoration a tenacious mucus, clear and slightly

pigmented. Temperature normal: pulse 104 to 112; weight of patient, nine stone.

I quote this case because I think it is as good an example of hæmorrhagic phthisis, in the sense of phthisis secondary to hæmoptysis, as is ever met with. There are three queries which must, however, be answered respecting it: 1st, Is it a case of phthisis at all? 2nd, Is it a case of phthisis caused by the inhalation of blood effused from the bronchial mucous membrane—phthisis *ab hæmoptoe*, in the sense of Niemeyer and others? or, 3rd, Is it a case of pulmonary hæmorrhage coincident with the occurrence of that active pulmonary congestion which is the very first stage of a certain number of cases of phthisis?

1. Though at present (November 17th, 1871,) there is no evidence of *wasting* of the lung, yet there is sufficient evidence of irreparable damage to its texture—*i.e.*, there has been slowly diminishing breath-sound, with abundant crepitation, since she first came under observation in September, until now the signs at the right apex seem only compatible with the air-cells there having become completely blocked with their epithelial products, now in the process of degeneration (caseous pneumonia).

The significance of such gradually developing apex-signs after hæmoptysis cannot, I think, be mistaken; softening and removal of lung tissue, to a greater or less extent, with indurative shrinking of the lung, are the almost necessary consequences. In all probability, the disease in the lower part of the lung is the direct result of the irritation of the inhaled blood, and much of it may no doubt yet clear up—indeed, had the patient obtained proper shelter and care from the first, it might have done so entirely; but, though all fever is now absent, the physical signs show that much of the lung yet remains clogged with inflammatory products.

2. I think the facts (*a*) of the opposite lung having wholly escaped, and (*b*) of the apex of the right lung being so deci-

dedly affected, are very strong evidence against the probability of the hæmorrhage having been from the bronchial mucous membrane.

3. The same two facts, and more particularly the gradual increase in the apex-signs, and the concurrence of the *copious* hæmoptysis with the severe lung-symptoms, are equally strongly in favour of the hæmorrhage being truly pulmonary—*i.e.*, of an acute pulmonary congestion, the true first stage of this (pneumonic) form of consumption having been attended with hæmoptysis to an unusual extent, the blood having also gravitated to other parts of the lung, and set up irritative changes there.

No doubt, at first—*i.e.*, soon after the hæmoptysis—the basic-were in excess of the apex-signs; this is the case in many instances of copious hæmoptysis in the first stage of phthisis. The explanation which appears to me most plausible is, that the natural effect of gravitation, aided by the expansive movements of the lung, is to remove the blood from the air-cells at the apex of the lung⁸⁶ which, therefore, often escape blocking, while they are the very physical conditions which most aid its entry into the lower portions. On the other hand, had the blood welled up from the base of the lung, it is unlikely, for the same reasons, that the opposite lung would have escaped.

It may be said—and here lies the uncertainty of all cases of this kind—that there was some disease existing at the right

⁸⁶ The blood sometimes coagulates too quickly for this, and then the physical signs vary greatly from day to day, as in an interesting case of Dr. Gee's, of which he has kindly shown me the notes. In this case the signs were *nil* after the first hæmoptysis: immediately after a second attack, however, there were signs at the right apex, which were modified from day to day. The hæmorrhages were frequently repeated here, and the blood expectorated was commonly dark and clotted, having apparently been retained some little time. Some temperature observations are much wanted immediately after the first copious hæmoptysis in cases presumed to be of hæmorrhagic character. Dr. Gee considers it to be elevated from the first.

apex prior to the hæmoptysis. It is true that the patient had some cough, but it was only very slight, unattended with constitutional symptoms of any special kind, and not apparently differing from the short cough so commonly associated with palpitation, of which she also complained.

We may say, at least, that the *onset* of the disease was *with copious hæmoplysis in a person previously with no apparent chest disease*, and, with the exception of some menstrual irregularity, and the palpitation so commonly associated with this condition, in fair health; we are further certain that *a considerable amount of the disease present is the result of the hæmoplysis*; and these two facts are sufficient to mark the case clinically as one of *Hæmorrhagic Phthisis*.

It is obvious that it is impossible to draw the line between those cases in which the acute congestion—whether of vicarious or mechanical⁸⁷ origin or arising from a chill or other cause—results in hæmorrhage in a lung previously sound, or determines what is often the first sign of consumption, hæmoptysis, from a lung whose vessels are frail from previous error of nutrition or disease at one portion. The distinction is mainly drawn from the clinical history of the case; and there is perhaps no true pathological difference, since we cannot on present evidence admit the hæmorrhage to be bronchial.

The chief fact upon which those who attribute early hæmoptysis to hæmorrhage from the bronchial tubes rely, is the extreme difficulty often experienced by the ablest auscultators in detecting any physical signs (but those, perhaps, of bronchial irritation) a short time after even very copious hæmop-

⁸⁷ The term mechanical source is meant to include those cases in which the excited action of the heart and pulmonary engorgement connected with violent muscular exertion—excessive dancing, rowing, etc.—lead to hæmoptysis. I do not know of any well-authenticated case of the kind. In Dr. Weber's first case (*Clin. Trans.*, vol. ii., p. 143), dancing evidently caused a recurrence of the hæmoptysis.

tysis. A man comes for examination a day or two after bringing up a large quantity of blood, and absolutely no signs which one could definitely pronounce as indicative of the origin of the hæmoptysis are discoverable. This very commonly happens. There may be the slightest comparative harshness and feebleness of respiration at the summit of one lung, from which long experience of the subsequent phenomena leads one to judge that the hæmorrhage has arisen there, but which without such experience would be considered wholly inadequate to account for the astonishing hæmorrhage.

I have already pointed out above that the readiness of escape permitted to the blood from the apex by the aid of gravitation and the expansile motion of the air-cells appears to me to be the explanation of this difficulty. It is the too common experience of the subsequent development of physical signs indicative of decided disease at the point which we could only guess to be the source of hæmorrhage before, which enables us in such cases to speak decidedly as to their nature upon what would otherwise be insufficient evidence.

It will be observed that the *hæmorrhagic diathesis* has not been spoken of in connection with hæmorrhagic phthisis. I believe, indeed, that the two diseases have no causative relation to one another. Cases are common enough in which there is a tendency to slight hæmorrhage from the gums, slight hæmoptysis apparently from the mucous membrane of the large bronchi or the throat, often associated with menorrhagia. I have watched many such cases for a long time, but none of them have ever become phthisical, or suffered from very copious hæmoptysis.⁸⁸ Cases of true hæmorrhagic diathesis are of course rare, and I am therefore glad to have the opinion of my friend Dr. Legg, who has paid much attention to *hæmophilia* and its literature, and who finds that the subjects of this disease are rarely affected

⁸⁸ This observation still remains true so far as my own further experience teaches me, *vide* chap. vi., for an account of this form of hæmoptysis.

with copious hæmorrhage from the lungs and that amongst them phthisis is still more rare.

Perhaps the chief advantage in retaining the term “hæmorrhagic phthisis” lies in its directing attention forcibly to the fact that hæmoptysis must not be looked upon only as the symptom or sign of disease, but as being also the *potential cause* of fresh disease. This consideration has a very important bearing upon the *treatment* of hæmoptysis.

We know that this early hæmoptysis is rarely fatal, and therefore, after calming the patient and securing for him perfect repose, we may anticipate spontaneous arrest of the hæmorrhage, or may endeavour to stop it by appropriate drugs and other means. Our whole anxiety is, however, in the immediate future, to watch for, and if possible to avert, the secondary consequences of the bleeding. Any detailed physical examination of the chest is, while the hæmorrhage continues, to be carefully avoided.

The thermometer, happily without danger to the patient, gives us the information we most require, and, together with the pulse and general aspect of the patient, is the best guide in the management of the case. If the temperature is raised at the time, or within a few hours of the hæmoptysis (it is often depressed for a few hours by hæmorrhage from the lungs), we may conclude it to be of congestive or inflammatory origin, and we anxiously watch for a few days to see whether the fever subsides with the hæmoptysis, or whether a fresh accession takes place significant of those secondary inflammatory changes we have reason to dread.³⁹

The patient is usually seen after the first burst of hæmorrhage; and if the bleeding continue after quiet is secured, astringents may be found useful. Liquid extract of ergot in

³⁹ Drs. Baümler's and Weber's cases, recorded in the *Clinical Transactions*, vol. ii., in which, however, they take a different view as to the source of the hæmorrhage, are most instructive in pointing out the value of the thermometer as a guide in the management of cases of hæmoptysis.

the dose of 3 j followed by smaller repetitions, and gallic acid similarly given in large doses are amongst the best astringents. Unless, however, active bleeding is going on, I am content in cases of hæmoptysis to give nitro-muriatic acid and ipecacuanha, the acid serving, I fancy, to give tone to the relaxed vessels which have yielded the blood.

The subsequent treatment of these cases requires the greatest care, and may be rewarded with brilliant results; for they are cases in which the disease is often in the smallest sense constitutional, and therefore in which recovery is always to be hoped for, while in no kind of condition is neglect attended with more unfortunate results than in hæmoptysis. I shall have a few more special remarks to make on the treatment of pulmonary hæmorrhage in connection with the subject of Recurrent Hæmoptysis.

The prophylactic treatment is of much importance when we have any suspicion of a tendency to pulmonary hyperæmia, especially in young girls before menstruation is thoroughly established, or if it be irregular. Violent exercise of any kind should be strictly forbidden, the underclothing should be of flannel throughout, and the air of the bedroom should be warmed.

CHAPTER VII.

Recurrent Hæmoptysis: Illustrative Case—main features of the disease; repeated copious hæmoptysis; chronicity of pulmonary disease—Pathology: slowly forming or old excavation, not necessarily tubercular; frequent absence of secondary fever; modes of arrest of hæmorrhage—Treatment, Prophylaxis—Both the forms of hæmoptysis described rare; significance of true hæmoptysis but little weakened by modern research.

THE following very typical example of recurrent hæmoptysis was under my observation for nearly five years and I will summarise my notes which have extended over the whole of this time, as briefly as possible:—

Thomas W., aged at the present date,⁴⁰ 31, described as a fitter, first came under my notice at the Brompton Hospital, in May, 1867. He had been ailing for some years with occasional cough, and had been three years previously under the care of Dr. Stone, at the same Hospital. He complained of pain in the chest and bad cough, but with, he said, no expectoration; he had had streaky hæmoptysis several times. He was doubtful whether he had got thinner; the appetite and digestion were good, the bowels regular, and the pulse slow. The only history of hereditary predisposition consisted in his father, an intemperate man, having died of consumption at the age of 44. The patient himself has always been a tolerably steady man of very active habits. He is very intelligent, of sanguine temperament, clear complexion, of medium height, and slight, though robust build. A striking feature about him, and worthy of note, is his extreme excitability—an almost superfluous energy with which he is gifted, which leads him to do everything with exaggerated effort.

At the date of his first attendance there was present at the left apex some dulness, with a few clicks.

On June 29th he reported having expectorated on the previous day a considerable quantity of blood, and, as he was still spitting some, he was ordered gallic acid powders.

On August 3rd he spat more blood, and *a note is entered of the existence of a small vomica at the left apex.*

8th.—“Hæmoptysis one pint this morning;” repeated powders, and ordered mist. acid. sulph. co.

On the 10th the hæmoptysis continued in a less degree and the breath was freer; he was ordered counter-irritation to the left apex and to continue the medicine.

On the 17th croton oil liniment was applied to the left apex. At this date, regarding the continuance of the hæmoptysis and its repeated occurrence at intervals together with the absence of any corresponding progress in the pulmonary physical signs, which were still limited to the summit of the left lung, I was first led to suspect the existence of a small aneurism of a pulmonary vessel there.

On the 24th, however, the hæmoptysis had almost ceased, and as the patient was emaciating, mineral acid and bark with small doses of oil were prescribed, and a linctus for the cough which was troublesome. With the exception of a very trivial attack he had no return of hæmoptysis, and ceased attending the Hospital, greatly improved in health, at Christmas of the same year.

He returned again in October, 1868, having continued, as he expressed it, “well” and at work until a fortnight previously. He had now slight cough and had expectorated some blood, but not so much as on previous occasions.

At this date there was “dulness on the left side anteriorly to the mamma, with high-pitched bronchial breath-sound, pectoriloquy, and cavernous cough; sounds very dry; some crepitus at angle of left scapula.” The oil was repeated, and an alkaline bitter ordered with small doses of iodide of potas-

sium. He again improved, having only one slight attack of hæmoptysis in November; and at Christmas, having an in-patient letter, was admitted into the Hospital under the care of Dr. Pollock, who confirmed the accuracy of the signs as above described. He only remained in a month, however, during which time I saw him on several occasions, and cautioned him against displaying so much energy in doing the most trivial thing, and coughing with such unnecessary violence. He had no appreciable expectoration, and left the Hospital feeling well.

I did not see him again until August, 1870, when, having remained quite well and at work until the previous Wednesday, he expectorated half an ounce of blood. The physical signs were still limited to the left apex, where there was dulness, bronchial respiration, crepitus, and friction (creaking pleura), and a whiffling murmur, systolic, audible in infra-clavicular region, continuous from subclavian not from pulmonary arterial region (and no doubt conducted subclavian murmur).

He ceased attendance in October, and continued pretty well until March 4th, 1871, when he again attended with hæmoptysis, and was seen by my colleague Dr. C. T. Williams in my absence, who ordered gallic acid immediately and directed him to send in three days' time. With his usual imprudence he attended personally on March 8th, having come from Battersea, though still spitting blood freely, and brought up a considerable quantity in the out-patient room. I prescribed gr. xx. of ergot every two hours for twelve doses, and ordered emp. lyttæ, four inches by four, to the left infra-clavicular region. This attack proved the most prolonged and desperate one he had yet had, and nearly terminated fatally.

March 11th.—Wife attended; hæmoptysis still continues; brought up half a pint of blood this morning at 2 a.m. Ordered six 3 ss. powders of gallic acid, one to be taken directly, and one-third (gr. x.) every two hours, with morphia linctus to allay the cough, and a brisk purge.

15th.—Hæmoptysis continues in a less degree. A mixture containing nitro-muriatic acid with glycerine and ipecacuanha (which I have found of great service at the close of an attack of hæmoptysis) ordered, and some more powders and purgative.

On the 22nd he had had three more attacks of copious hæmoptysis three days previously, and was extremely exhausted by the continual loss of blood. Sulphate of iron and alum were prescribed.

29th.—“Hæmoptysis half a pint yesterday; same amount to-day.” 3 ss. doses of gallic acid ordered every four hours for six doses.

On the 30th, mist. acid. sulph. co. 3 tis. horis; pil. plumb. c. opio gr. v., nocte maneque; iodine paint under left clavicle. From this date the violence of the attacks much abated—I should imagine rather from lack of blood-supply than from the efficacy of the remedies used, which, however, were steadily continued until April 13th, when he had had no hæmoptysis in quantity for a week.

On April 20th, some small doses of cod-liver oil were ordered and the acid ipecacuanha mixture with a little morphia. He had no more hæmoptysis after this, and again attended personally, though with great difficulty from his extreme weakness, on May 4th. At this date there was noted at the left apex “retraction of lung, dulness, cavernous respiration and rhonchus (slight).” Posteriorly there was “diffused crepitation, with some defective resonance.” This was the first occasion on which the lung had appeared to suffer from the effects of the hæmoptysis.

The cough was troublesome, especially in the morning, and on the 11th he was ordered ether and ammonia expectorant in the morning, lest his violent and unaided efforts at expectorating should lead to a re-opening of the broken arterial branch or possible aneurism, which seemed to have been the only conceivable source of such profuse and repeated hæmor-

rhage. It was extraordinary to note the rapidity with which the patient regained flesh, strength, and colour, though butchers' meat was only allowed every other day, stimulants were cut off, and abundance of milk alone permitted. He continued to take mineral acids and oil 3*j.* a day. He did not at all approve of the diet, but from previous experience of his rapid blood-making qualities I was convinced that a more generous regimen would have led to a return of the hæmorrhage.

On June 29, having only (on the 8th) had one comparatively slight attack of hæmoptysis, the physical signs showed enlargement of the right lung the margin of which reached across the median line; still some irritative bronchitis at the left base indicated by diffused submucous râles. Frictions with compound iodine ointment ordered.

Beyond an occasional tinge of the morning expectoration he has had no more hæmoptysis up to the present time, (December 1871) and has returned almost to his usual health, though the breath is shorter. Since June he has taken no oil; some digitalis was added to his mixture for a few weeks; and the diet has continued restricted, though less so of late.⁴¹

This case, the great length of which demands some apology to my readers, is one of extreme interest to me, as exemplifying well what I believe to be the main features of recurrent hæmoptysis, viz.:—1. Repeated copious hæmorrhages obviously arising from disease localized at one portion of the lung. 2. Pulmonary disease chronic in its course and but little influenced directly by the hæmorrhage. The hæmoptysis, though it may prove directly fatal, is accompanied by no severe fever or secondary pneumonia, and from it the patient frequently makes a speedy recovery.

⁴¹ 1876, I have seen this patient once or twice since, and quite recently in apparent health.

The pathological condition common, I believe, to all these cases of recurrent hæmoptysis is that of a slowly forming cavity, or one formed by a very localised process of an active character, in the walls of which pulmonary vessels still patent are exposed. I have seen some instances in which an aneurism has presented through the mucous membrane of a bronchial tube, or has occupied the whole cavity of a bronchial dilatation. It will be observed that the case above described did not begin with hæmoptysis; the man had had some dry cough and occasional streaky hæmoptysis for some years previously, and a few days after the first considerable hæmoptysis, a vomica was found at the left apex, where some two months previously there was consolidation and softening.

But the vomica, which yields the blood, need not be of "tubercular" origin—*e.g.*, a soldier was under my care at Brompton for fourteen months, who, in March, 1869, while blowing the clarionet, in India, was seized with hæmoptysis to the amount of about a quarter of a pint, which did not quite cease for about a week. A month or six weeks later, after having suffered for four or five days from severe pain and oppression in the right infra-mammary region, he suddenly brought up about a pint of "corruption" and some more blood, and since that time he had had hæmoptysis every few weeks. Whilst he was under my notice, the attacks of hæmoptysis were usually preceded by severe oppressive pain in the right mammary region. The pulmonary disease was mainly at the base or rather the middle of the right lung, there being scattered moist crepititation over the lung, with dulness, most marked at the base. Within the angle of the scapula, and also at the corresponding point in front, opposite the fourth rib, tubular respiration with some large click was heard.

This case appeared, then, to have begun with abscess in the lung—whether secondary to pulmonary apoplexy or not it

would be difficult to say—which had probably left behind a chronic deep-seated cavity.⁴²

The patient, Thomas W., whose case is above related, has never appeared to be febrile, and during the short time he was in the Hospital, on one occasion when he had hæmoptysis, though to a much less degree than usual—viz., one ounce—my friend, Mr. Bartlett, the Assistant Medical Officer, found his temperature to be normal.

In two other men subsequently under my care as out-patients, who, while in the Hospital, suffered from severe hæmoptysis, this same gentleman found no elevation of temperature—e.g., one case, James A., a stonemason (who had previously been under my care for some time with a vomica at the base of the lung, and induration at the apex, and who had several times had copious hæmoptysis while in the Hospital under Dr. Quain,) on November 14th brought up half a pint of blood at 10 a.m.

At 7.30 repeated half a pint, temperature 97.8°; 10 p.m. four ounces, temperature 99.2°.

15th.—10 a.m., temperature 98.8°.

5 p.m., temperature 98.2°, hæmoptysis three-quarters of a pint half an hour before.

7 p.m. and 9 p.m., temperature 98.2°. He had no more hæmoptysis, and the temperature, taken twice daily by Mr. Bartlett, up to the 21st, never rose above 98.4°. The physical signs were not altered, and the patient rapidly improved in general health.

⁴² He was discharged from the army, from Netley Hospital, with "abscess of the liver and phthisis."

Added Note: April, 1872.—No copious hæmoptysis for three months, but two or three ounces of currant jelly-like expectoration daily. No evidence of malignant disease. Signs of cavity very obscure.

2nd Note.—This patient came to see me in June of the present year, 1876, suffering from a recent bronchial catarrh. He had had no return of the hæmoptysis, and had lost his cough until two months previously. He had gained much flesh, and beyond some dulness and feebleness of respiration at the right base, presented no traces of his former malady.

In cases of fatal hæmoptysis, with very few exceptions, aneurism or erosion of a branch of the pulmonary artery has been found *post-mortem* at the Brompton Hospital. In some of these cases there had been previous attacks of hæmoptysis of the same character as the fatal one, while in several of them other vessels were found broken across, and occluded only at the very points of the fragments. Other observers (notably Dr. Rasmussen) have insisted on the frequency of pulmonary aneurisms in fatal hæmoptysis.

The danger in cases such as those I have described, is from the abundance of the hæmorrhage, which in a great number is the cause of immediate death on the first occasion. It is surprising this should not be so in almost all, and nothing is more striking than the recovery of some patients from what appears to be the most hopelessly profuse hæmoptysis—Nature apparently seizing the moment when, from faintness, the blood is at a standstill, to heal the breach by the formation of a coagulum. Hence the importance of withholding all stimulants till the latest moment.

Rokitansky refers to another mode of arrest of the hæmorrhage from a large vessel in a cavity—viz., by the cavity becoming blocked by coagulum, which thus compresses the vessel. I have seen an instance, *post-mortem*, in which the apex of the right lung was converted into a blood-cyst, quite closed, as large as a lemon, which had been produced by hæmorrhage into a cavity.

In the *treatment* of the form of hæmoptysis now under consideration, besides the general principles of absolute muscular rest, etc., before referred to, we must be more diligent with astringents and remedies which control the heart's action and allay cough: *ergot* acting upon the muscular walls of the arteries, *digitalis* diminishing the frequency of the heart's action, and *opium* lessening excitement and allaying cough, are of the greatest value.

Ipecacuanha emetics, admissible in certain cases of primary

hæmoptysis, would be certainly harmful in these. Our object is to allow the blood to coagulate at the seat of rupture, and faintness short of actual syncope should be encouraged, rather than prevented by stimulants. Nauseant remedies, however, from their relaxing effects on the vessels, are inadmissible. Interrupted cold applications to the chest may be tried in these cases more usefully, I think, than in those in which the hæmorrhage is capillary.

With reference to prophylactic treatment, patients the subjects of phthisis, particularly with chronic cavities, should be cautioned against muscular efforts, such as running upstairs or walking fast. The experiments of Colin⁴³ show that on exertion the pressure of blood in the pulmonary artery increases in greater ratio than that in the aorta. In those patients, too, who are gifted with rapid blood-making powers, and who pick up flesh with great rapidity after hæmoptysis, a timely partial abstention from butchers' meat, and the complete withdrawal of stimulants, may ward off or postpone the next attack.

I am inclined to think from my observation of the above related, and of some other cases, that after a greater or less number of recurrences of the hæmoptysis the vessel yielding the blood may become obliterated. Besides the cases I have already described, I may refer in further illustration of this point, to a very striking one, that of a middle aged man in one of Dr. Cotton's wards, who was admitted in June 1874. In the previous May and July he had had copious hæmoptysis (stated to be half a pint and over two pints), and throughout the months of June and July he had repeated attacks, sometimes two or three a day bringing up from four to six ounces at a time. The source of the hæmorrhage was apparently a large cavity in the upper lobe of the left lung. This man finally rallied and left the hospital at the end of six months much improved. He sought admission again in March 1876, his wife

⁴³ *Compte-Rendus*, p. 759, 1864.

having had a living child the previous year. He had had no more hæmoptysis when he came under my care. The right lung had considerably enlarged. He left the hospital again after two months treatment, the pulmonary disease having slightly advanced.⁴⁴ There is perhaps, however, a greater liability to the exposure and dilatation of a fresh vessel in such cases as have once borne the recurrent hæmoptysical character.

Having dwelt at considerable length upon two classes of cases in which copious hæmoptysis is a very prominent and important symptom—in the one class because it is the first symptom, which, though rarely directly fatal, is yet often attended with secondary results endangering the life of the patient; in the other, because the haemorrhage is always extremely dangerous, and may at any time prove directly fatal, while its secondary results are, as a rule, trivial, and but slightly influence the progress of the disease, which is usually one of the very chronic forms of phthisis—we must not omit to point out, for fear of misconception, that the cases which constitute these two classes are comparatively few and exceptional. Hæmoptysis, as a rule, whether very slight or moderately copious, is a merely casual, though very important symptom, in the course of phthisis.

I must, in conclusion, further state it as my firm conviction, that, take hæmoptysis from what point of view we may, its *genuine* occurrence in any degree beyond a mere streak in the expectoration, is a symptom the gravity of which, in the enormous majority of cases, has not been in the least exaggerated by the much-abused Laennec and others of equal experience—*i.e.*, so far as it is significant of positive disease.⁴⁵ Putting

⁴⁴ This patient is again (May, 1877) in one of my wards all active pulmonary signs and symptoms being in abeyance and his general health fairly good.

⁴⁵ Having redistributed the many diseases collected together by Laennec under his comprehensive term “tubercle,” we must beware lest, in our criticism of the symptoms attached by Laennec to the disease tubercle, we do not make sufficient allowance for our very restricted use of that term.

the matter in the most practical form, I presume there are very few Physicians who would venture to consider a candidate for life assurance as a "good life" who had the history of a distinct attack of hæmoptysis. But, on the other hand, by fully recognising the gravity of this symptom, and by, at the same time, bearing in mind the often-proved results attained by due precautions and improved treatment, we may justly give that very decided and conditionally hopeful advice which is most likely to meet with obedience and to be followed by corresponding success.

CHAPTER VIII.

Alveolar Catarrh before spoken of may set up local tuberculisation; why disease tubercular, not simple chronic inflammation; seat; progress independent of pneumonia; how different from miliary tubercle; mode of extension by continuous growth; clinical differences—Chronic Tubercular Phthisis the best clinical name for this disease—Relationship of local tuberculisation to local, miliary, and general tuberculosis one of degree or intensity as regards infective origin or specific constitutional nature—Distinctive characters of Chronic Tubercular Phthisis; prognosis—Sketch of a case of acute Tubculo-pneumonic Phthisis; distinguishing characters from Acute Tuberculosis and Acute Pneumonic Phthisis—General rule as to prognosis; necessity of watching the signs of fever as well as physical signs—Table representing chief varieties of phthisis, with their distinguishing characters.

In speaking of alveolar catarrh, it was remarked that, as in some cases it may proceed to catarrhal pneumonia of varying degrees of intensity, in which the pneumonic process with its intra-alveolar products gives the prevailing character to the disease, so in other cases the lymphatic or adenoid structures which so largely enter into the formation of the lung stroma, may, under the same primary catarrhal irritation, take on the more prominent growth: and great thickening of the alveoli, grey induration in which some individual granules of tubercle may or may not be distinguishable by the unaided eye, is the result—in fact, we have a local pulmonary *tuberculation*, of slower and more insidiously destructive progress than caseous pneumonia, so far as the lung is concerned, but more obstinately and continuously progressive, more prone to be succeeded by early implication of the other lung, supposing both are not from the first implicated, more quickly followed (sometimes even preceded) by disease in other organs particularly the larynx and intestines; and, in short, though a chronic or subacute disease, yet one of more early

average termination than the corresponding pneumonic forms of phthisis.

It will no doubt be said by those who only admit the existence of tubercle in the discrete or disseminately grouped miliary forms, that this *local tuberculisation* is no tuberculisation at all, that it is merely chronic inflammation. If so, it is an interstitial inflammation of a very special kind, that in typical cases spreads through the lung from apex to base, with a well-defined grey advancing margin, immediately beyond which the highly vascular but crepitant lung-tissue presents a striking contrast to it. On examining, however, more minutely with a lens, the alveolar walls are found considerably thickened to some little distance (perhaps half an inch) beyond the defined margin, though the alveolar spaces are not occupied with catarrhal cells—at least, not uniformly so, or to any material extent. I am not aware of any mere inflammation at all analogous to this in its invasive characters. It most resembles lupus of the cutaneous surface, which, I presume, no one would venture to describe or treat as merely inflammatory.

On the other hand, this form of tubercle (as I think it must be considered) differs from miliary tuberculosis pathologically by its primarily attacking one portion of one or both lungs (almost always the apex), and spreading therefrom, not by the dissemination of miliary tubercles far beyond the margin of advance, but by a continuous growth involving the destruction and subsequent excavation of the affected tissue. Clinically, the peculiarly insidious origin and progress of the disease with the gradually increasing *malaise* and anaemia, nocturnal cough, irregular fever, and the physical signs, at first very obscure at one apex, gradually increasing, and developing at the other, are in accordance with its pathology, and distinguish it also from the still more severe miliary form of tubercle. "Chronic tubercular phthisis" seems the best clinical name for this variety, of which the pathological pro-

cess is, as above stated, best represented by the term pulmonary tuberculisation.

Miliary tuberculosis, though not differing essentially in anatomical characters from the infiltrated form of tubercle above spoken of under the terms pulmonary tuberculisation, grey induration, etc., appears to differ somewhat in its mode of origin, which has been proved by late experiments and is now generally admitted to be *infective*. It may be, however, that we should better appreciate the relationship, so striking anatomically, which exists between these two forms of tubercle if we were to bear in mind that as there are two degrees of miliary tuberculosis—viz., first, that in which the tubercles are widely dispersed through many organs (infection through vessels?), second, that in which the granules are merely sprinkled around some old cheesy disease or induration (infection through lymphatics?)—so in a similar fashion a still less intense degree of infective power, amounting only to a specific irritation, may lead to the production of tubercle more limited still in extent, affecting only the parts contiguous to the infecting agent. Further, with a certain predisposition, it seems clear that both in animals and men miliary tuberculosis may arise from the mere irritation of a catarrh or local lesion, the specific quality being yielded by the constitutional peculiarity of the patient. And it may, correspondingly, be a question of degree of constitutional aptitude, whether the disease shall be as local as the irritation giving rise to it, or shall extend only by contiguity from the point of origin, or whether it shall at once take on the more virulent diffused miliary form. These observations are, I admit, in a degree speculative, but they surely find some support in clinical experience, and more from recent anatomical inquiries.

That some constitutional aptitude, hereditary or acquired, is necessary for the occurrence of tuberculosis is proved by its so often failing to arise in cases of chronic phthisis and scrofulosis in which for months and years inflammatory pro-

ducts in every stage of degeneration have existed as apparently efficient sources of infection; while in other cases the pathologist must explore with great diligence the body of one dead of tuberculosis to discover the required *cheesy mass*.

Returning now to the consideration of chronic tubercular phthisis, it is very difficult to depict in writing a case with sufficient accuracy to bring out those somewhat minute differences, the accumulation of which build up the distinction between the cases of catarrhal pneumonic phthisis already described, and those of chronic pulmonary tuberculisation to the pathology of which we have just referred. And indeed, it has already been pointed out that alveolar catarrh is really the first stage of both these diseases—or, rather, it is the true first stage of the one (pneumonic phthisis), and the determining cause of the other (chronic tubercular phthisis).

It will, then, perhaps be most instructive to enumerate those symptoms and signs the presence of which will warrant us in regarding the case as one of tubercular phthisis—as one, at all events, in which tubercle is the prevailing lesion; for it has been before stated that chronic tubercle is rarely wholly unmixed with other inflammatory products.

So far as temperature is concerned, there is nothing at present known characteristic of chronic tubercle. It is at times elevated, during which periods there are fresh accessions of disease, and the non-febrile intervals are of varying duration. In this respect the disease presents no important difference, so far as I have been able to observe, from the chronic pneumonic forms of phthisis. The physical signs are more characteristic. The obscure signs of alveolar catarrh do not give place to the well-marked dulness and coarse crepitation or crackling of catarrhal pneumonia, but to continued weakness of respiratory murmur, with impaired expansion or actual flattening, while moist sounds may be altogether absent, or one or two dry crackles may be elicited on cough. The percussion-note becomes hardened, and we may

suddenly be surprised by the appearance (having omitted to examine the patient for a week or two) of some feeble, blowing respiration, of hollow quality, still very dry, which increases in the same obscure way until an unmistakeable cavity is present. This formation of a cavity by a process of dry crumbling is very characteristic of the typical form of pulmonary tuberculisation.

Husiness of voice, or actual aphonia, is commonly one of the early symptoms in this variety of consumption, and is then, I think, characteristic of tubercle. The husiness may clear off, but the voice remains more or less altered permanently in quality. Too hasty a diagnosis must not, however, be made from this symptom, lest a grave prognosis be founded upon a simple laryngeal catarrh. The digestive organs are early affected ; the tongue presents a scanty white fur on a very red ground, with prominent red papillæ, an appearance which is very significant of intestinal lesion, still more so if the fur clears off in patches leaving raw-looking glazed surfaces ; and the symptoms characteristic of this lesion—alternating diarrhoea and constipation, with colicky pains, especially after food—soon appear.

Patients the subject of this form of phthisis are usually of slender figures and good features. Among them are those more interesting examples of consumption or decline that novelists prefer to describe. This variety is, however, much more uncommon than the pneumonic forms of phthisis.

As to prognosis, these cases admit of considerable temporary relief, and may appear to do well for the first few months. The physical signs progress however, and I think the duration may be pretty safely reckoned as within two years of the first appearance of definite signs. The intestinal or laryngeal complications cause great distress towards the last, and hasten the fatal termination.

The following sketch illustrates the phenomena characteristic of *acute tuberculo-pneumonic phthisis*—i.e., a case of pulmo-

nary tuberculosis, in which the tubercular granulations and groups of granulations are attended with much pneumonia, which latter is the main element destructive to the lung; while the former appears to stamp the disease with its peculiar adynamic characters, its continued fever, and determined progress without check to a fatal termination.

A woman, aged 31, was admitted into Dr. Cotton's ward at the Brompton Hospital in October 1871, and seen by me in his temporary absence. She had had "inflammation of the lungs" (there was no sign of old pneumonia *post-mortem*) two years ago, but had suffered from more or less cough, with frothy expectoration, for three years. Four weeks ago, however, she expectorated a small quantity, two teaspoonfuls, of blood, and the sputa continued to be tinged with blood for five days. She had since suffered from night-sweats, emaciation, cough, and pain in the side and between the shoulders, of which symptoms she complained on admission. The pulse was 112; the tongue furred; catamenia regular. She knew of no family predisposition to phthisis. The physical signs on admission were—harshness at the right apex, with subcrepitant rhonchus; at the left, jerking respiration and prolonged expiration. She lost rapidly in weight however, losing $3\frac{1}{2}$ lbs between October 26th and November 14th.

On November 8th the physical signs were noted as unchanged. On the 21st she was much worse, with a red tremulous tongue, a rapid pulse, great breathlessness, and much heat of skin. She was sitting up, but could with difficulty stand from the trembling of her limbs and weakness. Subcrepitant rhonchus was found diffused throughout the right side behind, with some defect in resonance not amounting to dulness. At my request Mr. Garton, clinical assistant, took the temperature night and morning from this date. During this time—twenty-nine days (five days the temperature was not taken) the maximum morning temperature was 103° , average 101.6° ; maximum evening temperature 104° , aver-

age $102\cdot3^{\circ}$; difference between the average morning and evening temperature $\cdot7^{\circ}$. On the frequent occasions when I saw the patient in the middle of the day the skin was uniformly hot, and the pulse very rapid—usually about 120. Meanwhile the pulmonary physical signs advanced, the crepitation became more abundant, and extended through both lungs. There were signs of breaking down at the right apex, though the presence of a cavity could not be with certainty ascertained. On December 19th there was present “diffused blowing respiration, with sonorous rhonchus and scattered crepitation more abundant at the bases, with some dulness; high temperature, and much dyspnœa.” On December 15th the patient began to be troubled with diarrhoea, which continued more or less to the last. The emaciation and loss of power rapidly increased, the smooth red tongue became white with aphthous patches, and she gradually sank, having never evinced, however, any delirium or other morbid brain symptoms.

The *continued* fever, the great and early prostration, and the diffused crepitation heard over the lungs, without any defined dulness, rendered the diagnosis of Pulmonary Tuberculosis being the prevailing lesion a tolerably certain one, while the absence of that degree of utter prostration with occasional muttering delirium, and the early presence of decided pulmonary signs, prevented one from regarding the case as one of acute general tuberculosis. There was, however, but little satisfaction to be derived from this reflection, for the prognosis was, so far as present knowledge could decide, inevitably fatal.

Post-mortem the lungs were found studded with racemose groups of tubercle surrounded by ill-defined areas of soft catarrhal pneumonia in active process of formation and degenerative softening; the right apex was breaking up into small cavities. There was no miliary tubercle on the pleural surfaces.

It will be observed that in this case there was a history of "inflammation of the lungs" two years before her fatal attack, and of three years' slight occasional cough and frothy expectoration. More or less long-continued bronchial catarrh is the very common precursor of pulmonary tuberculosis. It has before been observed that catarrh long limited to the bronchial mucous membrane may extend to the alveoli, giving rise to simple alveolar catarrh, catarrhal pneumonia, or tuberculosis, as something we do not yet understand shall determine.

There are, it is true, certain cases of acute pneumonic phthisis, "galloping consumption," in which the bronchopneumonia is so diffused as to render the diagnosis from acute tubercle at first very difficult; this matters little, however, for the prognosis is identical in the two kinds of cases. But the acute pneumonic phthisis, as a rule, proceeds by consolidation rapidly extending from one apex, or more rarely one base, with increasing dulness and abundant metallic crepitant sounds soon changing to gurgling. In such cases at the moment when the condition of the patient seems almost immediately hopeless the extension of the disease may stop, the temperature (more intermittent than in tubercle) may fall, and the patient may again begin to pick up strength while yet auscultation informs us that breaking down of the products of inflammation is still proceeding. Such cases point out what will generally be found a true guide to prognosis—viz., that the more prominently the symptoms of acute phthisis are attended with *defined* auscultatory and percussion signs, the more hope is there that the active disease may subside and the patient recover from the attack with so much damaged lung—in fact, the more certainly is the disease to be acute *pneumonic* and not acute *tuberculo-pneumonic* phthisis, or acute *tuberculosis*. Although, however, it is true that the disease, which we have watched progressing with such fearful rapidity as to lead us to anticipate death within a few days,

may stop short, yet the amount of damage its active extension has occasioned may be too great and the attendant systemic shock too severe for the patient ever to rally ; he succumbs at last to the exhausting influence of prolonged hectic after having lingered it may be for months.

It is imperative in these latter cases to watch closely, not the physical signs alone, for they will mislead us, but the signs of fever (temperature, tongue, and pulse), to learn the moment when the spread of the disease stops, and when tonic medicines, food, oil, etc., will have their due effect. Did we judge by the stethoscope alone, we should find softening and excavation signs increasing long after this period of true pause, because the products of the past inflammation must run through their normal stages of caseation, or liquefaction and removal, and these mere physical processes largely contribute to the production of auscultatory signs. Nor is it easy by physical diagnosis alone to say when the limit of the extension of the disease has been reached.

I may here, perhaps, usefully summarise in a tabular form the views advocated in the preceding chapters as to the distinguishing characters of the principal varieties of phthisis, which views have been acquired from the observation of a very large number of cases, and the making and supervision of many *post-mortem* examinations at the Brompton Hospital.

Table representing the chief Varieties of Phthisis, with their distinguishing Anatomical Characters.

ALVEOLAR CATARRH.

89

PHTHISIS.	PRIMARY LESION.	PATHOLOGICAL NATURE AND TENDENCIES.	ANATOMICAL CHARACTERS.	SUB-VARIETY OF PHTHISIS.
	Alveolar catarrhal pneumonia.	Terminates in recovery, or proceeds to catarrhal pneumonia. 1st degree terminates in resolution and recovery, or alveolar collapse. 2nd degree terminates in caseation, softening, excavation, or inturation from thickening and agglutination of alveoli. 3rd degree terminates in simultaneous degeneration and softening of alveolar wall and contents; ulcerative destruction. (α.) Copious haemoptysis may be coincident with the commencement of, and may perhaps determine, <i>pneumonic phthisis</i> , giving rise at least to secondary pneumonia.	Large cells of epithelial type, more or less blocking alveoli; greater or less degree of implication of alveolar wall, and proliferation of its elements.	Catarrhal pneumo-nic ph. (<i>progress</i> : acute, chronic, or intermitting).
PNEUMONIC.	Pulmonary capillary haemorrhage α.		Coagulated blood blocking alveoli, with (a) Hæmorrhagic ph.	
TUBERCULAR.	Pulmonary tuberculosis.	Of primary irritative origin, or part of general tuberculosis, or supervening upon caseous pneumonia or chronic tubercle. 1. Pure, terminates in death without breaking down of lung. 2. When mixed with pneumonia, caseation softening and ulcerative destruction of tissue. 3 (a) Irritative local grey induration, secondary to caseous pneumonia. (b) May be the primary disease progressively invading lung.	Nuclear growth of adenoid kind occurring as miliary grey granulations—(1) disseminated singly, or (2) in racemose groups, often associated with catarrhal pneumonia.	1. (*) Acute tubercular or tuberculo-pneumonic ph. (<i>progress</i> : acute, fatal). 2. Chronic tubercular ph. (<i>progress</i> : subacute or chronic, continuous).
FIBROID.	Pulmonary fibrosis	Present more or less in association with the chronic forms of the above varieties; never primary; when so marked in unilateral cases as to give a special clinical character to the disease is conveniently named separately.	Contractile fibroid cicatricial tissue, the results of proliferation of elements of fibrous stroma of lung, including vessel sheaths and lymphatic tissues, mingled with products of primary disease. Aneurismal dilatation, and recurring symptoms in the more chronic indurative forms of phthisis, attended with quiescent or slowly forming cavities.	Fibroid ph. (<i>progress</i> : very chronic; with some times long quiescence). Ph. with recurrent hæmoptysis.
	Pulmonary arterial haemorrhage. β.	(β) Copious haemoptysis may be a marked symptom in the more chronic indurative forms of phthisis, attended with quiescent or slowly forming cavities.		

* Acute tuberculosis cannot be regarded as a variety of phthisis, though it frequently supervenes as a fatal complication of the disease. Simple Inflammatory Phthisis.

CHAPTER IX.

LARYNGEAL PHthisis.

Laryngeal Phthisis, term used to signify those cases of Phthisis in which the Larynx is early or very prominently involved. Pathology of the disease—Morbid Anatomy. Symptoms and signs. The use of the Laryngoscope. Diagnosis from Alcoholic or irritative Catarrh, Syphilitic Laryngitis, Functional Aphony. Treatment.

It sometimes happens in the adult that phthisis is ushered in with laryngeal symptoms, and the variety of the disease thus arising ranks amongst the most fatal and distressing of all.

Strictly speaking the term laryngeal phthisis should be applied only to those cases in which the laryngeal lesion is primary, but in practice it is more loosely employed to include all those cases in which laryngeal symptoms constitute an early and a striking feature. And the latter employment of the term is not only more convenient, but is also more in accordance with the general pathology of the disease; for although undoubtedly in many instances the disease of the larynx is the first local manifestation of phthisis, yet in all cases the lungs speedily become involved, so that to attempt more rigidly to restrict the term would imply what is not the fact—that there is a phthisis which begins and ends with laryngeal disease. Laryngeal ulceration, indeed, most commonly of all arises as a complication in advanced pulmonary phthisis, and in these cases even grave pulmonary lesions are often forgotten in the greater urgency of the throat symptoms, so that they are grouped under the heading of laryngeal rather than of pulmonary phthisis. Under this heading then let us, without further regard to logical appropriateness, consider that form of laryngitis which is a part of, or which may complicate, phthisis.

As regards the pathology of the disease authorities differ much, some regarding it as a primary or secondary tuberculosis of the larynx and trachea in which points of tubercle appear in the submucosa, and rapidly becoming caseous and softening produce minute ulcers, which, by their coalescence form larger ones, whose walls and floors are the seats of fresh tubercular deposits. Other pathologists consider that the lesion commences as a scrofulous catarrh, or inflammation, of the mucous membrane which speedily involves the submucous glands and determines in some of them caseation softening and ulceration. I must confess that my own observation would lead me to adopt the latter view as much more generally in accordance with clinical and *post-mortem* finding. I do not doubt, however, that true tubercular disease of the larynx does now and again occur. Curiously enough the only case of "tubercular" ulceration of the larynx that has been published in the *Pathological Society's Transactions* within the last ten years, was one in which the disease appeared to be truly tubercular. In this case which was exhibited by Dr. Wickham Legg, the laryngeal disease came on after tracheotomy (performed for croup) and, co-incidentally with it, there was miliary tuberculosis of the meninges. I have myself failed to meet with anything like a miliary tuberculosis of the laryngeal or tracheal mucous membrane. Tubercle growths speedily form however about the scrofulous ulceration, adding as with the pulmonary and intestinal lesions the characters of inveteracy and destructive spread. Persons in whom these lesions occur are certainly highly predisposed to phthisis, but it is a remarkable fact that laryngeal phthisis is exceedingly uncommon in children. My experience may be exceptional, but I can only recall to mind one well-marked case of phthisis in a child, a little boy aged about six, in whom the disease commenced in the larynx. Nor do laryngeal symptoms commonly occur in children in the course of phthisis. These are my principal reasons for thinking that "tubercular" laryngitis

only rarely arises as a primary or secondary *tuberculosis* of the larynx, but that it does commence rather in accordance with the most common beginning of the disease in the intestines, and even in the lungs, as a scrofulous catarrh whose products are very prone to decay and to soften, causing ulceration and tuberculisation around them. On the plane surface of the membrane these results do not happen, it is in the follicular recesses, and in the gland structures, that caseation of inflammatory products ensues.

The morbid appearances found *post-mortem* in the larynx consist of a more or less general inflammation, and inflammatory thickening of the mucous membrane of the larynx, and upper part of the trachea with points or patches of ulceration. The membrane is as a rule hyperæmic, and of a bright red colour in the early stages; this colour, which fades somewhat with death, is retained in patches of minute vascular injection, especially in the upper trachea. In more chronic cases the membrane becomes more or less dusky or slate coloured. In the earliest stage soft and œdematous, the membrane soon becomes thickened and the œdema of a more solid kind, these characters being especially marked in that portion covering the arytenoid cartilages, and in the aryteno-epiglottidean folds; the membrane covering the whole or at least the under surface of the epiglottis is also usually thickened, and the action of these parts is stiffened and impeded by the induration surrounding them. The favourite seat of ulceration is the base of the arytenoid cartilages; a ragged ulcerous hole is often found in this situation at the bottom of which the denuded and roughened cartilage may be sometimes felt. The point where the vocal cords meet in front, is also a frequent site for deep ulceration, sometimes extending through to the subcutaneous cellular tissue. The mucous membrane above the false cord and over the inner surface of the true cord at its posterior termination is commonly also attacked, and in the more advanced stages all these parts may be in-

volved together. The trachea, especially at its upper portion, and the under surface of the epiglottis, are often the seat of ulceration spreading from minute points and giving a peculiar worm-eaten look to the surfaces, *vide* Plate I. The cartilage of the epiglottis is not in this form of laryngitis directly attacked as is so frequently the case in syphilis, but it may lose vitality, and become necrotised in points from deprivation of its mucous membrane. The shallow ulcers below the glottis may also become extensive and deep causing extensive exposure of cartilages; such an appearance, however, is suggestive of syphilitic complication. The great number of glands dotted over the epiglottis and the trachea explains the peculiar character of the ulceration in both these situations.

The sufferings of the victim of laryngeal phthisis are great and varied. One of the first symptoms that attracts attention is an alteration in the tone and quality of the voice. It becomes husky and usually deepened in tone. Vocalisation is uncertain, the voice sometimes failing into a husky whisper, to come out again, on increased effort being made in speaking, with a reverberating deep tone. In some cases, however, even at the earliest stage, the voice is suppressed from an inability to approximate the cords. The patient suffers some oppression of breathing, which may amount to serious dyspnoea, the respirations and the pulse being both quickened. He looks ill, loses flesh, and suffers from sweats and evening fever. A troublesome teasing cough of a harsh dry character attended with but scanty and difficult expectoration, which gives rise to some pain and increases a sense of rawness in the throat, is complained of. Some tenderness is elicited on deep pressure in the upper tracheal region, and a small tender gland or two may be felt in the hollow between the margin of the sterno-mastoid and the larynx. Pain on deglutition is a frequent symptom, and it is still more characteristic when described as shooting upwards to the ears causing in them "pricking sensations." As the

disease proceeds and the ulcerative destruction of the larynx becomes more extensive, the aphonia becomes complete, and the cough most distressing and paroxysmal. At this period the lung disease has usually proceeded to excavation, and the expectoration is more abundant: at the same time effective cough is almost impossible, in consequence of the patient being no longer able to bring about its essential condition, closure of the glottis. Deglutition is also most difficult and painful from the irritable and stiffened epiglottis failing effectually to guard the laryngeal aperture, and from the pharyngeal muscles compressing the painful and swollen parts.

The fauces and tonsils may be natural looking, they are often pale and drier than natural with their small sub-capillary veins too well marked. The back of the pharynx is frequently granular looking, sometimes even abraded, with streaks of viscid mucus adherent to the surface. The mucous glands are too prominent. A distinct ulcer may sometimes be seen at the back of the pharynx, or behind one tonsil having the characteristic raised, irregular margin, and granular ash coloured surface of the tubercular ulcer.

An ulcer in rare cases may be present upon the tongue, or on the inside of the cheek especially about the orifice of the parotid duct on one side. Such cases are by no means necessarily syphilitic although careful inquiry on this point should always be made. On deeply depressing the back of the tongue with a spatula the epiglottis may in some cases (those in which deglutition is painful,) be seen to be red and inflamed.

But, as I have already observed, the throat so far as it can be seen by the unaided eye, may be perfectly natural, it is so perhaps in the majority of cases. A view of the disease can only be obtained by means of the laryngoscope, and a few words must here be said as to the best means of using that instrument in the diagnosis of these diseases.

It requires no more skill in the use of the laryngoscope than can readily be gained by a little perseverance and practice, to recognise the principal diseased conditions of the larynx, whether these be paralytic or inflammatory, ulcerative or trophic. In certain cases, however, in which operative interference is needed for the removal of growths or which are attended with peculiar difficulties, more special skill is requisite. With these latter cases I do not venture to deal, but must refer my readers to the admirable special treatises which have been published on the subject.

For ordinary practice, all the permanent apparatus needed is a suitable reflector⁴⁷ with throat mirrors of two or three sizes.

Sun-light is the best when it can be obtained. A "Silber" or "Duplex" lamp will yield a sufficient light, but the most suitable of artificial lights (excluding the oxy-hydrogen and other lights which are not easily obtained) is that derived from an argand gas burner, concentrated by a bull's eye condenser.

The patient should sit upright well back in a chair, opposite to, and at a little higher level than, the observer, so that he is within comfortable reach. The light should be placed close at the side of the patient's head, at about the level of his ear, and other lights should be excluded from the room. If sun-light be employed the patient should sit somewhat obliquely with his back to the window. The observer now arranges the mirror upon his forehead so that reflected light will be projected to the back of the patient's throat; he should then direct the patient to open the mouth and put out the tongue, but *not* to hold the breath as most patients try to do during an observation. A towel having been adjusted round the end of the tongue, it should be gently pulled forward by the left hand, whilst with the right the laryngeal mirror, previously warmed, is introduced. The laryngeal mirror should be introduced (without touching the tongue) with its back to the soft palate, so as to bear back the uvula, and bring the reflecting surface in a vertical line with the glottis whose image it reflects. The condition of the epiglottis and of the vocal cords and their surrounding parts is now seen, and between the cords a view may be obtained of the interior of the trachea.

By directing the patient to say *ah—h—h* the vocal cords will be observed in action, and any defect of a paralytic kind affecting the vocal muscles can be observed; by directing him to breathe deeply the respiratory movements can be noted. If the epiglottis be pendulous, so as partially to hide the

⁴⁷ The reflectors adapted to a spectacle frame with a ball and socket joint with laryngeal mirrors, are supplied by Messrs. Mayer and Meltzer, of Great Portland Street, in accordance with Dr. Morell Mackenzie's directions.

cords from view, a gentle traction of the tongue may suffice to elevate it, or by making the patient say *ch—h—h* the glottis is drawn upwards usually distinctly into view.

There are many little practical difficulties to be overcome which render a satisfactory examination of some patients difficult or impossible with ordinary skill.

Among the most important precautions and expedients to be adopted are the following :—

1. Before taking any further steps, have the light and the patient perfectly arranged so that you can quite comfortably project a good stream of light to the back of the pharynx.
2. Take the tongue gently between the thumb and finger and be very careful to avoid dragging it down upon the teeth.
3. Avoid touching the back of the tongue in introducing the mirror, or retching will be caused.
4. If, as is often the case, the throat be very irritable, desist for a time, and let the patient sip a little iced water before proceeding again.
5. If the patient be timid, let him or her hold out the tongue for you, and do not attempt at first to get a full view of the larynx.
6. If after two or three fair attempts, the throat be still too irritable to permit a view of the larynx, do not persevere further, but make another examination at a future time.

With the aid of the laryngoscope we can thus see at least, quite a sufficient sample of the morbid conditions present, to make certain our diagnosis and to prompt our treatment. Acute or chronic catarrh, inflammatory oedema, and thickening and ulceration at one or more points may be observed. The epiglottis often appears thickened and rigid, but we cannot usually see ulcerations upon it, for when present they are as already stated, generally limited to its under surface. Its margin is, however, sometimes roughened or furrowed-looking. Thickening and oedema of the aryteno-epiglottidean folds is most often to be observed, and is considered by Dr. M. Mackenzie to be most characteristic of this disease. In the earlier stages an angry looking spot or two of ulceration may be seen often over the arytenoid cartilages, of a yellowish white colour shining out from a red, tumid, smooth, inflamed ground. Ulceration in the more deeply seated parts may, or

may not be seen, but intense injection of the mucous membrane is often visible. In cases of more advanced date, the orifice of the glottis is seen to be distorted by ulceration and thickening, the cords being involved in the havoc so as to render their approximation impossible. In this stage especially, much purulent secretion may collect in the ventricles and about the cords.

The *diagnosis* of tubercular laryngitis rarely presents any real difficulty. The diseases which most simulate it are chronic alcoholic, or irritative catarrh, syphilitic laryngitis, and hysterical aphonia. The absence of fever, the presence of a definite exciting cause, and the laryngoscopic signs of general catarrh without local thickening or ulceration, are usually sufficient to distinguish the chronic catarrh arising from dust or drink, from laryngeal phthisis. Alcoholic catarrh is moreover always associated with a similar affection of the pharynx. Syphilitic laryngitis is more difficult to distinguish from tubercular, and the two diseases are sometimes combined. Besides the general distinguishing features of syphilis, the local characteristics are larger ulcerations with a less degree of thickening. The ulcers are also more sharply cut than in phthisis. Extensive destruction of the epiglottis is pathognomonic of syphilis.

In functional aphonia the aspect of the patient, the absence of fever and the whispering character of the voice, are in themselves sufficient for diagnosis, sufficing to show a defect of will and not of function. The mobility of cords is not impaired in this affection, at least in its earlier periods, nor does one see how anything like a paralysis of vocal muscles could be rectified by one shock of electricity.

It sometimes happens, however, that in the early stage of tubercular disease, when there is as yet no discoverable ulceration, the voice is suppressed completely. With the laryngoscope the cords are seen not to come in apposition, in fact they appear to be partially paralysed. The impeded

action may be the result of stiffness from the thickening present, but I suspect it is also partly nervous, from the depressing effect of the pulmonary disease reflexly affecting the larynx through the pneumo-gastric. This early suppression of the voice is most likely to be confounded with hysteria, it occurs in nervous people but the lungs are in all cases more or less involved.

Although the *treatment* of the laryngeal disease in phthisis is confessedly very unsatisfactory and in many cases hopeless, yet we can certainly do much to lessen suffering. In the earlier stages an even temperature with steam inhalations and warm poultices, with warm milk and seltzer frequently to drink will give much relief. We have, indeed, in all cases, two conditions to treat, laryngitis and laryngeal ulceration: the latter, having once occurred, being the abiding lesion, the former (laryngitis) being more or less intense at different times and sometimes subsiding altogether. As in the lungs and in the intestines, so in the larynx, it is the attendant inflammation that causes the chief symptoms and calls especially for treatment. Now and then the local application of a strong solution of nitrate of silver (40 grs. to the oz.) or of sulphate of copper (20 grs. to 1 oz.) is useful. The former especially sometimes lessens sensibility of the parts. But I am quite sure that in this disease too great a diligence in the use of local applications to the larynx is to be deprecated. I have not found much benefit result even from the use of astringent solutions in the form of sprays. But such sedative or slightly stimulating inhalations as hyoscyamus, tincture of benzoin, hops, &c., are often very useful, and I have found great advantage from the employment of the ipecacuanha spray recommended by Dr. Ringer and Mr. Murrell for bronchitis. When there is much pain in the larynx, the persistent use of counter-irritation will be found often most beneficial. A small blister the size of a shilling should be daily applied over the region of the larynx for several days, so as to keep

up constant but not too severe counter-irritation. This treatment may be combined with sedative inhalations, and rarely fails to give considerable relief, even in hopeless cases. Where the difficulty and pain in swallowing are great, nutrient enemata should supplement, or for a time wholly replace the ordinary method of feeding ; they give rest to the diseased parts and lessen the terrible sense of exhaustion from which the patients often suffer.

The dyspnœa in these cases never becomes urgent enough to suggest the expediency of tracheotomy which on other grounds would not be thought of.

CHAPTER X.

ON PULMONARY CAVITIES.

Pulmonary cavities.—Why separately considered: their presence does not necessarily constitute the third stage of phthisis—*Recent cavities*, their formation, physical signs indicating caseous abscess—Expectoration, significance of elastic tissue in expectoration the same as that of humid crackling râle: neither necessarily significant of present activity of disease—Extension of cavities—Rindfleisch's views—Symptoms: fever, chills, night sweats, expectoration—Treatment: Quinine, arsenic, salicylic acid, salines—Inhalations and cough-mixtures rather to be avoided—Sputa should be expelled—Recent cavity may cicatrise, or become quiescent and contract: its bronchus may become obliterated—*Scarcting cavity*, description of, signs and symptoms—Albuminoid degeneration of organs, diarrhoea—Objects for treatment. 1. To lessen secretion. 2. To evacuate what secretion is formed. 3. To disinfect—Counter-irritation, inhalations—*Active or ulcrous cavity*, description of, signs and symptoms—Treatment—Inhalations, support stimulants, ammonia and bark or iron, counter-irritation—Local treatment of cavities.

THE excavation stage of some cases of phthisis is so prolonged and the symptoms are so decidedly grouped about the cavity, that at some schools it is the custom to name such as cases of “cavitation,” Whilst I do not think such a term admissible in any formal sense, however useful it may be as a colloquial expression in clinical teaching, there are yet many points in the diagnosis and treatment of different kinds or conditions of cavities which I think may form useful matter for consideration in a separate chapter.

We have seen that destruction of lung is the essential anatomical feature of pulmonary phthisis. In the most rapidly fatal cases the destruction takes place simultaneously at many centres, or involves such a large extent of lung as to render hopeless any effort at repair or compensation. In the major-

ity of cases of phthisis that come before us, however, the disease as already pointed out affects principally one apex, the active symptoms attendant upon the pulmonary consolidation and softening after a time subside, the appetite returns and the patient begins to gain strength and flesh. The cough still continues, however, and auscultation reveals the existence of a cavity at the apex concerned; the disease being now usually described as having advanced from the first (consolidation) through the second (softening) to the third (cavity) stage. If these terms were strictly employed in a structural or anatomical sense as regards the lungs only, they would not be objectionable; but in fact they are too often extended in their application to the phthisis of the patient and therefore become fruitful of error and misunderstanding. These so-called stages of the whole disease phthisis have reference merely to the effects of that disease upon, perhaps, a fiftieth part, or perhaps, nearly the whole of one or both lungs; they have no meaning as applied to the present or prospective duration of the disease. A man with a big cavity is very frequently infinitely better off as regards life and health prospects than one with a "first stage" patch of disease no larger than the area of a shilling. A cavity once formed is so much lung gone, and it is for many reasons much better that the irremediably diseased portion should be cleared out than that it should remain as a centre for fresh irritation, or which may break down or infect the system at any time. Our anxiety as regards the immediate prognosis rests upon the condition of the out-lying portions of the affected lung and still more upon the degree of integrity of the opposite lung. Yet the student rarely looks beyond a cavity, upon the discovery of which, in accordance with current phraseology, he classifies the case and decrees the fate of the patient. The physician too is often nowadays beset by anxious inquiries from the relatives and friends of his patient, as to the existence or non-existence of a cavity, upon which they base their hopes and fears, and upon

his capacity to discover which, his reputation is registered in their estimation! These terms then, being inaccurate and misleading, should never be used in their general sense.

To resume, however, the special subject of the present chapter. Cavities may be considered under four heads: 1, the recent cavity; 2, the quiescent cavity; 3, the secreting cavity; 4, the active or ulcerous cavity.

Recent cavity—The recent cavity is the first result of the breaking down of caseous nodules in the lung. Whatever the derivation or constitution of the pulmonary consolidations may be, they, in cases of sufficient intensity, undergo degeneration in few or many centres, and liquefaction of the caseous products ensues. We do not, however, get any physical sign of this liquefaction or of the production of a cavity until communication is effected with a bronchus and some of the softened matter is expelled. From this moment we have cavities existing in the lungs and accessible to the air during respiration.

To yield the auscultation signs which are regarded as necessary for diagnosis, a cavity must have the dimensions of a walnut or larger and must communicate freely with a bronchus. But on comparing our clinical notes with post-mortem observations we shall find the former most commonly inadequate if we have awaited the presence of cavernous breathing and pectoriloquy and such like orthodox signs before admitting the existence of excavation.

The pulmonary consolidations break down into cavities in one of two ways which are not, however, essentially different. Firstly, many minute lobular centres of softening may arise which at first yield to auscultation moist crackling or humid clicking sounds, which increase in size and abundance as the softening centres extend and coalesce into larger cavities, until finally we get cavernous râle. The respiratory murmur which—bronchial with the first consolidation—had become weakened and more or less masked by the moist sounds becomes again audible but much altered in

quality, now assuming the more or less distinctly cavernous character. As the cavities become fewer and larger by coalescence the other well-known signs of cavity become apparent—pectoriloquy, splash on cough, *bruit de pöt-félé*, &c.

Perhaps the large majority of phthisical cavities form and increase in the way I have thus briefly sketched, but in some cases they are formed in a slightly different manner. It not infrequently happens that we fail to get distinct evidence of pulmonary softening for some time after troublesome, but more or less dry, cough and hectic symptoms point very strongly to its presence. There may be dulness, harsh breathing, and some fine spongy crepitus, increased after cough, but none of those distinct clicks characteristic of pulmonary softening. Then the patient will suddenly, in the course of the night perhaps, expectorate a considerable quantity of purulent matter, and we find evidence—cavernous rhonchus, &c, of the existence of a cavity. The explanation of these phenomena is obvious enough, a nodule of consolidation of appreciable dimensions, but rarely exceeding a walnut in size, becomes uniformly caseous and then softens in its centre and gradually liquefies throughout before communicating with a bronchus, when its fluid constituents are at once expelled, and auscultatory evidence of the existence of a cavity becomes abruptly developed. In the *post-mortem* room we may often cut through such softening nodules in all stages of ripeness for exit, they sometimes undermine and rupture through the pleura, and may well be designated *caseous abscesses*.

Prof. Rindfleisch⁴⁸ ingeniously explains the manner in which these softened masses finally communicate with the corresponding bronchus. He observes that there is a general traction upon the branches of the bronchial tree during inspiration, and that as soon as the softening process has sufficiently advanced, a separation is effected between the root

⁴⁸ Ziemssen's Cyclopœdia of Medicine.

of the softened mass and the bronchus which passes into it. Air is immediately drawn into the rent, and the liquid contents of the cavity escape through the bronchial tube. I have satisfied myself that the caseous abscesses above described, do probably discharge themselves by a process of this kind.

With the softening of the pulmonary textures the expectoration ceases to consist of mucus, it is no longer viscid, tenacious, and more or less frothy, but contains opaque specks and purulent streaks, and gradually becoming more purulent each sputum is moulded in its escape through the air-passages, to form a more or less isolated nummular mass. Sometimes, especially as the cavities increase in size, the sputa become more diffluent. As the expectoration increases in abundance, it becomes also easier, and the patient describes his cough as being looser, but soon complains of the amount of expectoration.

At the earliest period of commencing excavation a careful examination of the sputa will discover the presence of elastic tissue in it. The discovery of elastic tissue in the sputa has indeed almost exactly the same significance as that of the physical sign of moist crackling, and in all doubtful cases it is well that evidence obtained by the microscope should confirm the stethoscopical signs. Even the most skilled auscultator will often be glad of the help of this instrument. For this purpose the expectoration should be collected for twenty-four hours, and then boiled with an equal quantity of solution of caustic soda, (20 grains to the 1 oz.) with frequent stirring, the boiling fluid should then be poured into a conical glass vessel and freely diluted with distilled water. A more or less copious sediment deposits, numerous specimens from which should be diligently searched with the microscope for the boldly curved loops and fragments of pulmonary elastic tissue, Figs. 2 and 3. If any be found we have absolutely sure evidence that removal *i.e.* excavation of lung

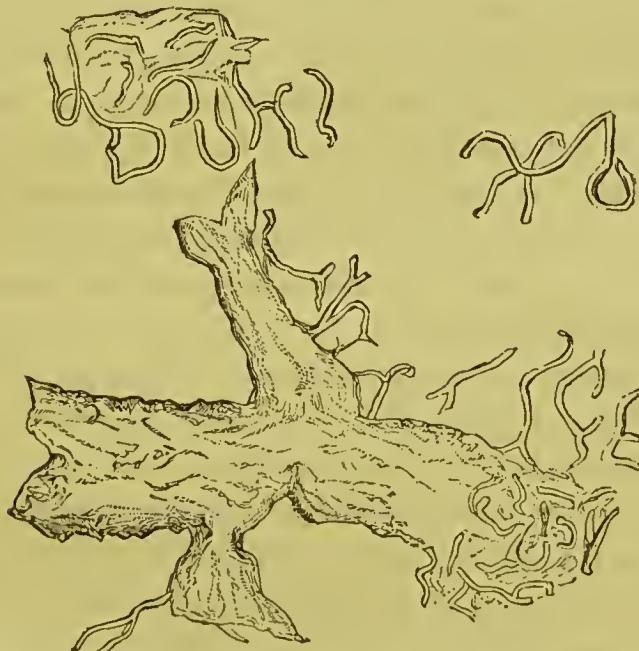
FIG. 2.



Elastic tissue and fragment of small vessel, from expectoration of patient with rapidly-forming cavities.—Drawn by Dr. Sydney Coupland. $\times 200$.

is proceeding. Fragments of vessels, or minute bronchi may also sometimes be recognised in the sputa, Figs. 2 and 3.

FIG. 3.



Elastic tissue and fragment of bronchus (?) obtained from expectoration of patient with active pulmonary softening.—Drawn by Dr. Sydney Coupland. $\times 200$.

Just, however, as I have already more than once remarked,

that the physical sign of moist crackling may be produced by the degenerative liquefaction of the products of a *past* inflammatory affection, so the presence of elastic tissue in the sputa does not *necessarily* signify present activity of disease, but simply the removal of its caseous effete products.

A cavity of recognisable dimensions having formed, it may extend indefinitely by new solution of tissue, and by the coalescence into it of smaller cavities, or it may cease to extend. In the latter case it may continue to secrete much purulent fluid for a long time, or it may become quiescent and undergo more or less contraction. The ordinary method of extension and enlargement of cavities by softening down of fresh pulmonary tissue into the original cavity, and by the merging of adjacent smaller excavations into one larger one requires no further comment. It is obvious that all trabeculated cavities have been formed in this way, the trabeculae being the remnants of the septa which formerly separated the smaller cavities from one another. There is, however, a theory of Prof. Rindfleisch's by which he explains the enlargement of bronchial and other cavities, which I must notice here although I think there are many and important objections to its acceptance. He regards the obstruction of numerous small bronchi by the pulmonary consolidations as necessitating an increase during inspiration of the air pressure upon the interior of the bronchi in front of the obstruction, and also upon the interior of cavities, thus leading to their dilatation. Prof. Rindfleisch conceives that the soft walls of recent cavities readily yield before this increased air pressure, and thus enlarge towards the pleural surface condensing the tissue around them. When we consider, however, that the influx of air into the lungs does not take place in any constant quantity, but awaits the aspiration dependent upon the expansion of the thoracic cavity, we see that this theory cannot apply to the enlargement of phthisical cavities nor even to ordinary cases of bronchiectasis. For in both these morbid states, but especially in phthisis, the lung is

more or less consolidated or thickened, and having its pleural surfaces adherent, at least over those portions which are excavated, resists expansion more than in health. In vigorous people with healthy lungs the utmost available inspiratory force only exceeds that necessary to expand the lung by from 2 to 3 inches of mercury. In phthisis this reserve force is much diminished, the vital capacity of the chest is much lessened, so that we have less air entering the lungs and at less pressure. Hence I think the inspiratory force, effective in producing certain forms of emphysema, has no appreciable action in dilating pulmonary cavities. I have not observed in phthisis except in advanced cases with marked dyspnœa any excessive effort with inspiration, the muscles of inspiration lessen in vigour with the general wasting. During cough however, the intercostal spaces over a superficial cavity become noticeably bulged, and with the stethoscope we may hear the air forcibly rushing into the cavity, so that doubtless the repeated cough has a tendency to dilate cavities somewhat, but even this is only an auxiliary force in effecting their enlargement.

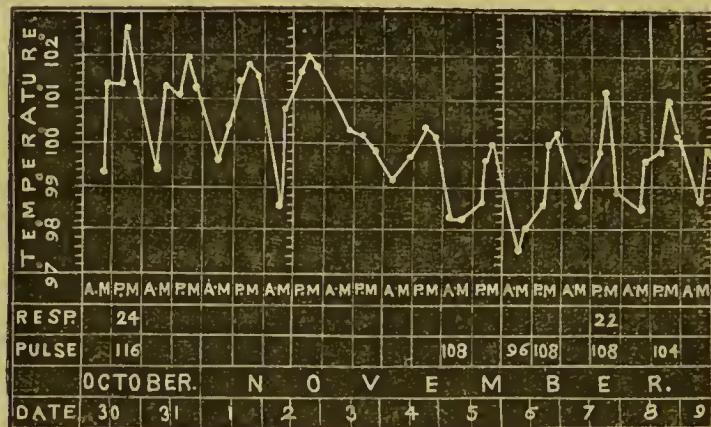
Whilst cavities are forming or extending, the symptoms that present themselves for *treatment* are fever with wasting, anorexia, profuse expectoration and sometimes severe haemoptysis.

The temperature chart of a case of phthisis with recently formed and extending cavities depicts a markedly hectic type of fever. The temperature mounts up to a considerable height, from 101° to 103° in the course of some hours during the day, the maximum temperature being usually attained at some period between 2 p.m. and 10 p.m. and from this point subsiding to a point below the normal, the sub-normal range occurring usually towards the early hours of the morning. This range of temperature normal to the period of active cavity formation in phthisis has been well illustrated by my colleague Dr. C. T. Williams.⁴⁹

⁴⁹ *Medico-Chirurgical Transaction*.—Vol. lviii., 1875.

It is sufficiently indicated for clinical purposes by observations taken two or three times a day, provided we are careful to note the period of the day at which the fever is highest and to record a daily observation at that time. We must be further careful to remember that a normal morning temperature means, in these febrile cases, a sub-normal early morning temperature as this has an important bearing on treatment. The first half of the subjoined chart, Fig. 4, shews very fairly

FIG. 4.



the type of fever under consideration, it is taken from a case of actively extending excavation, and represents a much longer period of observation.

Morning or afternoon *chills* are exceedingly common during this period of phthisis. They rarely amount to rigors but during some fixed period of the day for from half an hour to two hours the patient feels chilly and miserable. These chills are rarely followed by sweatings.

Night-sweats are not infrequently complained of in these cases, most generally so indeed in cases not under treatment.

In cases of recent and active pulmonary excavation the principal symptoms we have to take into consideration, viz., fever, night-sweats, cough and expectoration, are for the most part dependent upon the local pulmonary lesions.

It can scarcely I think be doubted and indeed it is generally

admitted, although no sufficient proofs of the fact have yet been adduced, that the fever at this period of phthisis is mainly dependent upon some form of purulent absorption. The often very decided effect of remedies points to the same conclusion, for they are remedies—quinine, arsenic, salicylic acid—of an antiseptic kind.

The usefulness of *quinine* in these cases is generally appreciated, nay, it is in my experience too favourably regarded. But if well borne it will in moderate doses, combined with a little mineral acid, help to sustain the patient and to lessen night sweats. This method of treatment is as a rule far superior to the older plan of attempting to reduce fever and clean the tongue with salines. In some cases, however, disordered digestion or diarrhoea requires special attention.

Arsenic cannot be given in these cases in sufficient doses to diminish the temperature in any striking degree but it has a marked influence upon some of the most distressing symptoms attendant upon the fever and it seems sometimes in a remarkable manner to improve the general condition of the patient. This drug is most indicated in those cases in which daily recurring chills are complained of. Three to five drops of the Liq. Arsenicalis or the Arseniate of soda solution, or $\frac{1}{2}$ of a grain of the Arseniate of Iron three times a day, will often suffice to entirely prevent the recurrence of these chills. Arsenic has also been extensively used for this purpose by my colleague Dr. Pollock, and generally with good results.

Salicylic acid is a drug which has a marked effect upon the temperature of phthisis and especially at the period under consideration. In the case whose temperature is depicted in the above diagram the drug was commenced on the morning of the 3rd of November, and the immediate fall of temperature is well shewn. This case is, however, by no means a favourable one of its kind to illustrate the good effects of the drug; but it is, perhaps, additionally useful in serving to shew when its action fails as well as where it succeeds. The case was

one of extensive excavation and softening proceeding with relentless energy, the larynx was also extensively, although not at the moment actively, involved.

My notes including careful temperature observations by my friend and assistant Dr. Blackader extend over a period of ten weeks during seven of which the patient was taking more or less constantly the soda salt of Salicylic acid.⁵⁰ The decided fall of temperature on the first administration of the medicine in 20 grain doses every four hours is very marked, but after a few days the temperature is observed to rise again somewhat, although not to its original height. This happened on several occasions after the drug had been omitted for a few days, and we observed the same to happen in other cases of equal severity. In cases, however, of less extent of lesion the fall of temperature has proved enduring. Another very marked effect noticed during the administration of this drug, not only in the case referred to but in two other cases of equally fatal severity, was that although the fever was not controlled and the lung disorganisation certainly advanced, yet the strength and weight of the patients were remarkably maintained. Thus the weight of the patient referred to in the diagram, a man 5 ft. 4 in. in height, was a week after admission 7 st. $12\frac{1}{2}$ lbs. A fortnight later it was also 7 st. $12\frac{1}{2}$ lbs. He now (Nov. 3rd) began the salicylate and on Nov. 21st he weighed 8 st. 6 lbs. During this period the temperature had ranged at first 2° then 1° lower. On Dec. 5th he weighed 8 st. 4 lbs., having for the last four days taken quinine in small doses instead of salicylate and suffered a slight increase of temperature. Again on the 16th of Dec. he weighed 8 st. $6\frac{1}{2}$ lbs. the salicylate having been recommenced Dec. 6th with 1° reduction of temperature range. He then gradually but slowly lost weight but on leav-

⁵⁰ 20 grs. of this salt with $\frac{1}{2}$ to 1 dram of Syrup of Lemons and min. 5 of Tinct. Chloroform comp. in an ounce of water make a not unpalatable mixture. Some patients prefer the drug in an aromatic water.

ing the hospital Jan. 13th with all the physical signs increased he still weighed one pound heavier than on admission.

It is quite obvious that in all these cases now in view, there is to be taken into account, not only the softening down of caseous products, and suppurative processes in the walls of recently formed cavities, the symptoms of which, we think, may be referred to purulent infection, but also fresh consolidation and caseation proceeding, over which it is doubtful whether, save in an indirect way, antiseptic drugs have any influence. It is not intended to suggest that salicylic acid or any other drug can cut short the progress of Phthisis or Tuberculosis.

Another symptom in diminishing the severity of which salicylate of soda has an influence, is *cough*. My clinical assistant, Dr. Blackader, first noted the fact that the amount of expectoration and the cough were diminished by this drug, and I can confirm his observation. The drug may be detected in the sputa (it is abundantly excreted by the urine) and hence it is probable that it may have some local action upon the cavity wall as a disinfectant.⁵¹

Amongst the unhappy effects of salicylic acid, must be named its physiological action which is attended with noises of bells in the ears, deafness, and sometimes great depression of spirits. The temperature is apt to become depressed below the normal, so that it is often desirable only to give the drug during certain periods of the day.

Beyond the assuring a fair amount of sleep during the night, the use of sedative cough mixtures should be avoided. We certainly do not desire to encourage the accumulation of the highly irritating and septic products of caseous solution. On the other hand, I have not found inhalations of much avail, and am doubtful that they may do harm by increasing the exercise of injured tissues. When we examine

⁵¹ I would venture to suggest it as well worthy of trial in cases of bronchorrhœa.

post-mortem the walls of recent cavities still bounded by as yet unliquefied caseous material we see how impossible it is to hope for healing effects upon the cavity walls in this stage. There is a further and a last point to which I will refer in regard to this active stage of cavity formation, and it is with respect to the importance of *expectorating* the products of pulmonary softening and cavity secretions. Many people, and especially delicate minded females and sensitive men, feel a delicacy or a timidness in expelling the products of their disease. Children often do not know how to do so. This inability or unwillingness to bring away the sputa is a fruitful source of stomach and bowel troubles and possibly of local tuberculosis.

A cavity having formed may at once cicatrise, or may become quiescent and contract or cicatrise, or may continue secreting indefinitely.

(a). In a certain number of cases, cavities, and especially those having their origin in caseous abscesses, *cicatrise*. Laennec describes this as the natural mode in which the pulmonary lesions healed, and seems to regard phthisis as incurable in the first stage. Taking the term cavity in its strict sense, as meaning any loss of pulmonary substance, however small, cicatrisation is probably common, nay, we find *post-mortem* evidence of the cicatrisation of cavities large enough to come within clinical recognition, but unfortunately they are usually surrounded by other cavities and disease centres which continue to enlarge and render the cicatrisation of one amongst them of little avail.

I will here quote a case which I brought forward for examination at one of a series of clinical lectures delivered at the Brompton Hospital in January, 1877, as illustrating what I regard as a rare phenomenon, viz., the rapid cicatrisation of a cavity at the apex, arising from caseous abscess, and the complete clearing up of all physical signs and symptoms.

"A. W—, a pale, scrofulous-looking girl, has been under my observation at this hospital since March, 1872,

when, at the age of fifteen, she first came to me as an out-patient. She had then a somewhat loud cough, which had troubled her all the winter, but there were no other definite symptoms and no discoverable pulmonary signs. Her family history was good, but she had been delicate since an attack of measles in childhood. She got well on ordinary treatment, but returned in October, 1874, and again improved, but was ailing during the winter. In the spring of 1875 her principal complaint was of a painful affection of the left breast; she still had no definite pulmonary signs or symptoms beyond general delicacy and slight cough. In June, however, she had whooping-cough rather severely; and six weeks afterwards she still whooped with the cough, which was attended also with thick difficult expectoration. On now examining her chest again I was somewhat surprised to find dulness with very marked cavernous breathing and some gurgling below the right clavicle, and moist crepitant râles were rather sparsely scattered over the posterior base. Fever and hectic symptoms were now marked. The prognosis appeared grave, and I recommended her to obtain an in-patient's letter. She did not come into the hospital, however, until January; meanwhile she improved as an out-patient. In October the sounds were noted as being quite dry, and on her admission into my ward in January, I examined her chest with great care, and did so repeatedly afterwards, but I could never discover more than some harshness and feebleness of breathing at the right apex.

I take it that this was a case of caseous abscess at the right apex, which cleared out and cicatrised. Such cases are, however, unfortunately exceptional; this is the only one of the kind of which I have a definite and certain note. Other similar cases have, however, been reported, and *post-mortem* observation of cicatrices in the lungs leave little doubt that they occur more frequently than positive clinical experience would indicate; but they are rarely single and uncomplicated,

as in this case. One feature in which this case differs from others which I shall show you at a future meeting is in the absence of any marked flattening or displacement of organs."⁵²

(b). It more commonly happens that a cavity having attained certain dimensions, becomes *quiescent*, all progress in the pulmonary disease being arrested, its walls become condensed and toughened by the development of fibrous tissue, so as to shut it off from the surrounding lung. The contents of the cavity become less and less abundant, the sounds yielded to auscultation more and more dry, until no moist sounds at all are heard even on deep breathing, but only a few clicks and a characteristic succussion of air are produced on the patient coughing. This kind of cavity at once begins to shrink somewhat in size, its walls becoming denser and thicker by a cicatrical process. The chest wall flattens and the heart and opposite lung encroach towards the affected side, to make up for the loss of space. If the surrounding lung be tolerably sound it will become expanded around the cavity, so that the latter if only of moderate size and not very superficial may become altogether obscured. It is very common *post-mortem* to see a longitudinal wrinkle or fold upon the surface of the lung bounded by expanded vesicular tissue, and on making a vertical section through such a wrinkle, we cut across a more or less deeply seated cavity which has evidently undergone contraction. But even superficial cavities may become in this way lost to clinical observation. There are in the Brompton Hospital museum, some examples of a condition which is well but rudely depicted in Laennec's work.⁵³ A tough fibrous band dips into the surface of the lung, and is surrounded by radiating wrinkles between which the pulmonary vesicles are expanded. The band is firmly attached by one end to the summit of a contracted cavity situated half or three quarters of an inch below the surface, and by the other intimately at-

⁵² Lecture II.—*Lancet*, Jan. 27th, 1877.

⁵³ Sir John Forbes' edit., 1827, Plate v, Fig 2.

tached to the costal pleura. This band in fact represents an adhesion which originally existed over a superficial cavity but which has become lengthened by the contraction of the cavity and the knuckling inwards of the lung to form the wrinkles described.

That even large cavities may cicatrize and become permanently obliterated is a fact ascertained by *post-mortem* observations, but a close attention to the subject convinces me that this is of very rare occurrence. It is, however, quite common for the physical signs of a cavity which has undergone a certain degree of contraction, to disappear, being replaced by simply suppressed or very feeble (conducted) breath-sound. This arises, however, not necessarily from the cavity becoming obliterated but from the bronchus with which it communicates becoming narrowed or occluded by the dense cicatricial growth in the cavity-wall, in which cicatricial growth the sheath of the bronchus partakes. Such a cavity although it may perchance communicate with a few collateral minor bronchial tubes is practically or completely *closed*, and this is the next best thing to its being obliterated. It diminishes in size and ceases to take any further part in the production of pulmonary symptoms.⁵⁴

Whilst a cavity is drying up the cough becomes irritable and the patient frequently complains of its "tightness" having been accustomed to easy expectoration whilst the secretion was abundant. He should be encouraged by explaining to him the favourable nature of the cause of his difficulty, and

⁵⁴ I have not infrequently observed clinically the complete loss of all signs over a cavity of considerable size, and their return after a few days, shewing that a temporary closure of the bronchus had taken place probably from a plug of mucus. Probably the periodically abundant and foetid expectoration found in some cases in which there can be discovered no signs of cavity is due to a cavity still secreting but only communicating obliquely with a bronchus, so that the secretion becomes pent up for some time before it can find an exit.

directed to check so far as possible by an effort of the will the tendency to violent cough. Sedative cough mixtures, injurious whilst the expectoration was abundant, now become of much value. More or less morning expectoration persists for a considerable time after the cough has ceased during the day. The further medicinal and climatic treatment of cases of this kind must be conducted on general principles.

(c). The *secreting cavity* is usually a cavity of tolerably old date which has ceased to extend and is unaccompanied by active pulmonary disease. It is a dense-walled cavity like the last which is lined with a smooth opaque pyogenic (false) membrane, which can be readily scraped off exposing a highly vascular, dusky red, subjacent surface. The trabeculæ, which are numerous, present the same vascular surfaces, and false membranes. Such cavities may go on indefinitely secreting a diffluent creamy pus, they yield gurgling sounds, with marked amphoric breathing, and dull tubular percussion. Then there is either no fever present or it is trivial, consisting of a slight rise of temperature only at night. The tongue is clean with a tendency to redness and loss of epithelium. Although the appetite usually continues good, the patient slowly loses ground, and acquires the sharp hungry features peculiar to chronic phthisis, with clubbing of the fingers and toes, and a tendency to albuminoid degeneration of organs. Diarrhœa is apt to supervene, and troublesome sickness is sometimes occasioned by the cough. These cases are always of a precarious kind and are not very amenable to treatment.

The objects we have in view in the treatment of such cases are:—1. To lessen secretion. 2. To promote evacuation of what secretion is formed; and 3. To disinfect such cavities.

Counter irritation is useful over the cavity in the form of strong iodine applications, or flying blisters, or perhaps a blister kept open for a time by savin ointment. Acids and astringent iron tonics with cod-liver oil are needed. Sedative

cough mixtures are directly contra-indicated save at bed-time and then solely for the purpose of procuring rest. It is in these cases that inhalations are most useful, for,—firstly, there being no actively spreading disease present, they are not contra-indicated: secondly, we can, by their use, render less noxious the pus that bathes the surfaces of the cavity and is apt to become inhaled into distant parts of the lung: thirdly, there can be little doubt that appropriate inhalations sometimes have a healing or alterative effect upon the internal surface of the cavity. The best substances for inhalation in these cases are, Iodine, (vapour iodi BP.) occasionally and for a short time: Carbolic acid, (glycerine of carbolic acid 3*i.*—3*ii.* to half-a-pint of hot water): Tar water (Liq. carbonis detergens 3*i.* or 3*ii.* to half-a-pint of hot water.) They may be taken very well from a deep jug or a Nelson's inhaler with the sponge removed. Perchloride of iron or other astringents may sometimes be used with Siegle's spray apparatus, but I have myself hitherto been disappointed with the effects of atomised astringents in pulmonary diseases, and doubt if they penetrate so far as vapours ordinarily inhaled.

In favourable cases the secretion dries up and the cavity becomes quiescent.

Unfavourable cases slowly lose ground from albuminoid disease of other organs, or from recurrent diarrhoea. Also, besides the tendency in all cases of phthisis for fresh lung to become involved from a distinct attack, there is in these cases a danger, not commonly recognised, of some of the abundant secretion becoming inhaled during cough into lower parts of the same, or of the opposite, lung, and thus setting up fresh centres of disease. The infra mammary region on the opposite side is a favourite seat for this secondary disease to appear.

Hæmoptysis is not common from either the quiescent or the secreting cavity, but it sometimes occurs in a dangerous and unexpected manner from the rupture of an ectasia or aneu-

rismal dilatation projecting from the unsupported cavity side of a large pulmonary vessel.

Ulcerous cavity.—The last kind or condition of cavity I wish to refer to is formed in the usual way and may have been *quiescent* or merely *secreting* for some time when, from exposure to cold or to erysipelatous influences, or other causes, it assumes a state of active ulcerative extension. Such cavities are angry-looking, deep dusky red on their inner surfaces, often studded with hæmorrhagic points or ulcerative erosions, they are highly trabeculated and very irregular in shape, but sharply demarcated from the lung tissue by a thin vascular wall. They contain a copious blood-stained purulent secretion which, when expectorated, is mixed with the ropy secretion from the intensely vascular bronchi which communicate with them. The lung tissue surrounding the cavity is injected and œdematosus, and at distant parts of the lungs may be found pneumonic centres, which evidently owe their origin to the inhalation of the acrid secretions from the cavity. In such cases there is sharp fever present with quick pulse, furred tongue, and a tendency to typhoid symptoms. The expectoration is usually mixed with blood, or dark changed clots may be removed from the cavity. Sometimes copious hæmorrhage takes place from the erosion of a large vessel.

These cases are always immediately dangerous and are usually unfavourable. They may best be combated by quinine internally, or in some instances, by full doses of perchloride of iron, and by sedative inhalations, (tincture of benzoin and opium, hyoscyamus and chloric ether, carbolic acid and opium). Ipecacuanha wine administered as spray by Siegle's inhaler is well worth a trial in this condition of cavity. The patients must be well supported, and have abundance of good air, for these cases are probably nearly allied to erysipelas. I have known them to be endemic in a ward which was overcrowded, and to have ceased on a bed being removed therefrom. It cannot indeed be too carefully

remembered in the treatment of phthisical affections of the lungs that such patients have internal *wounds* or *sores* which, unlike most other internal affections are accessible to the contamination of foul air, and that erysipelatous processes may be readily set up in them which are apt to be recognized only as *intercurrent pneumonias* or other local inflammations.

After the more active general symptoms have lessened, if the blood-stained, and copious expectoration lead us still to infer that the walls of the cavity are hyperæmic, if not ulcerated, I am convinced that the best treatment will be found to be free counter-irritation. A blister should be applied over the site of the cavity, and should be kept freely discharging for several days by means of savin ointment dressing. I have seen the active local symptoms completely subside under this treatment, and the expectoration from being abundant and sanguineous, become scanty and viscid, apparently consisting of bronchial mucus only.

I have, as yet, said nothing about the local treatment of cavities save by vapour or spray inhalations. But the treatment of pulmonary cavities, and even of pulmonary consolidations, by puncture and injections through the thoracic wall, has been attempted by several physicians. Nor do I think that their efforts, albeit in some instances ill-timed, merit the hasty and indiscriminate censure of the late Dr. Hughes Bennett, who concludes a sweeping condemnation of all measures of local treatment whether by inhalations or otherwise, with the following remark;—“The result of all these efforts has been—what an intelligent consideration of the pathology of the disease might have anticipated,—a uniform failure.”⁵⁵

Dr. F. Mosler⁵⁶ of Greisswald relates three cases of cavities in the upper part of the lung, two of which he injected with solution of permanganate of potash through a fine Dieulafoy's

⁵⁵ Reynolds' *System of Medicine*. Vol. iii. p. 589.

⁵⁶ Ueber locale Behandlung von Lungenkavernen von Dr. Fr. Mosler Prof. im Griesswald.—*Berliner Klinische Wochenschrift*. Oct. 1873.

syringe. In the third case he introduced a "thick silver drainage tube" which he covered with carbolised lint and through which much purulent secretion escaped. In this case, on the subsequent occurrence of haemoptysis he injected through the tube a weak solution of perchloride of iron. It does not appear that in these cases the patients were injured by the treatment. Dr. Wm. Pepper of Philadelphia⁵⁷ has also treated three cases, and in one case with, he believes, positive benefit. His plan is to employ local anaesthesia, and the patient having taken a full breath, to introduce a Dieulafoy's No. 1 (finest) needle. He then injects four to ten minims of diluted Lugol's solution (*liquor Iodi. PB.*) in the proportion of four min. to 3 i water. Dr. Pepper suggests that "superficial circumscribed indurations or caseous infiltrations of the lung tissue may have introduced into them through fine needles such injections as may tend to induce absorption or reparative action." This last suggestion is very unlikely to be carried out with any good result, indeed its adoption seems on every ground unjustifiable. The principal value of the experiments above named, and of others which have been made is to shew that, if necessary, lung cavities can be, with due precautions, injected with astringent or disinfecting fluids.

The two questions that now arise are—(1.) Is it ever necessary or advisable to adopt this method of treatment? (2.) Can we inject into the lung astringent or other fluids in sufficient quantity or with sufficient frequency to be of any practical usefulness? I have no experience of my own to offer in answer to these questions, nor do I think they can at present be answered. I have never myself, however, seen a case of apex excavation that I should in the least feel tempted to treat by the "local" method. But there are cases of rare occurrence, (I have at least one in my recollection but which was not under my sole control,) in which a large old

⁵⁷ *Philadelphia Medical Times.* March, 1874.

cavity situated at the extreme base of one lung and secreting profusely, gives rise to hectic, to very troublesome paroxysmal cough attended with vomiting, and at the base of the opposite lung may be detected patches of lobular pneumonia, which have evidently arisen secondarily from inhalation of purulent matter derived from the cavity. When I meet with such a case again I shall certainly take into earnest consideration the propriety of introducing a drainage tube into the cavity, and of employing astringent injections. Whereas in apex cases any injected fluids have a tendency to gravitate to lower portions of lung, in the basic cases such as that above referred to, there would be no such danger.

Amongst the complications that may arise during the breaking down of the pulmonary tissue in the formation of cavities, there is none so grave, or so well meriting a fuller consideration than it has hitherto met with, as Pneumothorax, to which I propose therefore to devote a separate chapter.

CHAPTER XI.

PNEUMOTHORAX.

PNEUMOTHORAX.—Laennec's views, controverted by Jaccoud—More commonly arises in course of phthisis than from other causes—May occur at any period—Mode of origin, (a) from sphacelus of pleura, (b) from fistulous perforation.—Composition of gas—Symptoms and signs vary with valvular or non-valvular nature of opening—Summary of symptoms. Physical signs—Hydro-pneumothorax—Percussion resonance, auscultatory phenomena, and cardiac displacement more fully considered—M. Gaide's views—Mechanism of displacement of heart. Case illustrative of—Dr. Hayden's views—Intra-thoracic pressure—Experiments showing effect of collapse and compression of lung upon circulation through it—Diagnosis of pneumothorax—Prognosis—Treatment.

PNEUMOTHORAX has since the time of Laennec,⁵⁸ who first rendered it clinically recognisable, attracted considerable attention especially amongst French and English writers. Laennec described pneumothorax as of three kinds, viz., simple pneumothorax, pneumothorax associated with fluid effusion into the pleura, and pneumothorax with fluid effusion and a fistulous opening communicating with a bronchus. In the first two non-perforative varieties of pneumothorax, the gas found in the pleura was, Laennec affirmed, effused there by simple exhalation from the pleura (or the fluid it contained), or it was the product of decomposition. In the third or perforative variety the air gained access to the pleura through the fistulous opening in the lung. In an able article in the *Gazette Hebdomadaire* for 1864, M. Jaccoud⁵⁹ maintains that there is no sufficient proof of the occurrence of simple pneumothorax, and expresses serious doubts as to the occurrence of air in the pleura at all, save occasionally perhaps from the decomposition of liquid effusion.

⁵⁸ *Traité de L'Auscultation*, T. ii, p. 240. et seq.

⁵⁹ *Gazette Hebdomadaire*, 2ieme Série, T. i. 1864.

M. Jaccoud contends that the cases of so called simple or essential pneumothorax, have probably been cases in which minute perforation of the pleura has occurred, the aperture having soon closed, and, in some instances, the air having undergone absorption. There can be little doubt that M. Jaccoud's criticism is just, and that although in gangrenous and some allied conditions of the lungs or pleura, some gaseous products of decomposition may collect in the pleural cavity, or the air may, as suggested by Dr. Gairdner, transude through the more or less devitalised membrane, yet such cases are uncommon and are moreover rendered unworthy of notice beside the gravity of the lesions which have given rise to them. Hence we may safely hold that perforation of the pleura is the only important cause of pneumothorax.

The rupture of an air vesicle in emphysema, or the separation of a slough in gangrene, or the bursting of an hydatid, or a haemorrhage through the pleura, may be enumerated amongst the occasional causes of pneumothorax. One of the most common forms of perforation is that which arises either from bursting of an empyema through the lung, or its artificial or natural escape through the thoracic wall. Setting aside such, however, as not being cases of genuine pneumothorax, and excluding gunshot and other wounds of the thorax or lung from without, we find that perforation of the pleura from within most generally occurs as a complication in phthisis or pulmonary tubercle.

The constant breaking down of tubercular and pneumonic infiltration in the course of pulmonary phthisis tends, by undermining the pleura and interrupting its vascular supply at certain points, to cause it to soften and to give way during some trifling increase of air pressure from cough or other cause. The observation of this constant tendency of the pulmonary lesions to bring about perforation of the pleura in phthisis led Dr. Scott Alison⁶⁰

⁶⁰ The physical examination of the chest in Pulmonary Consumption 1861, p. 270, *et seq.*

to regard this accident as worthy of being considered the fourth or perforative stage of the disease, a view which has, however, not met with general acceptance.

Perforation of the pleura may occur at any period of phthisis: even at the very commencement of the disease a small subpleural tubercle may soften and break through the pleura, but such an occurrence is much more common in the more advanced stages. In the majority of cases of phthisis as already pointed out, pleurisy of a dry and adhesive kind accompanies and keeps pace with the progressing lung consolidation. But it may be observed *post-mortem* in some cases, which are marked by acuteness of process, and by the pneumonic character of the consolidations, that no adhesion between the pleural surfaces has taken place: each pleural surface, but the visceral pleura more especially, being covered by a thin finely granular semi-transparent layer of lymph which may readily be scraped off with the knife, leaving the shining, almost healthy-looking pleura denuded. These are the cases in which pneumothorax is especially apt to occur. At certain points of the surface of the pleura thus affected will be seen opaque yellow spots, of a size varying from that of a pin-head to a split bean, one or two of which may have already given way, and on making a vertical section through any one of them, we divide a more or less softened caseous nodule having its centre perhaps already excavated. The relationship of the bronchus communicating with the subjacent cavity whether it open directly or obliquely into it, and the nature of the opening through the pleura itself, whether it be free and direct, or valvular, give rise to most important modifications in the signs and symptoms which follow upon the rupture.

Pneumothorax is thus most likely to happen in the more acute pneumonic forms of phthisis. It is least apt to occur in the more chronic fibroid forms of the disease. In these latter varieties of phthisis, however, the complication is sometimes met with. In some papers on this subject contributed

to the Medical Times and Gazette, for January and February 1869, I pointed out that sinuses, in no respect differing from the sinuses one sees extending from chronic abscesses, or carious bone, may sometimes be found extending from old cavities towards the surface. Such sinuses may penetrate both layers of the pleura and open into the subcutaneous cellular tissue of the thoracic wall, or if, as is rarely the case, the pleura be not firmly adherent, a communication with the pleural cavity is established. In one remarkable case described in the paper referred to, a sinus had opened from an old cavity through the posterior mediastinum into the opposite pleural cavity. When a communication is thus effected between a more or less deep seated cavity and the pleural surface, the perforation is so oblique or sinuous as to be always practically valvular.

Pneumothorax is most common on the left side. This is in accordance with the experience of Louis, seven out of the eight of whose cases were left-sided; and of Walshe, who has collected eighty-five cases (thirteen of them observed by himself) fifty-five of which were left-sided. Out of sixteen *post-mortem* observations I have found the pneumothorax to be on the right side in five cases, on the left side in nine cases, and in one case double-sided: in the latter case, however, the perforation on the *left* side and consequent effusion of fluid had preceded for some months the rupture of the right lung. Again including the above in a larger series of 39 cases culled from the Brompton Hospital records, the number of left-sided cases is 23, that of right-sided cases 15. Hence *post-mortem* observation, whilst still giving the preponderance to the left side, tends more to equalise the frequency with which the two sides are affected. Out of these 39 cases, 27 were males, 12 females. (*Vide* Table pages 142 and 143).

The gas effused into the cavity of the pleura approximates in composition to that of expired air. Dr. Davy⁶¹ found 100 parts to consist of carbonic acid 8 parts, and of nitrogen 92 parts.

⁶¹ *Philosophical Trans.*, 1823-24.

In five examinations of the gas from a case of pyo-pneumothorax at different periods he found it to vary in composition, the highest proportion of carbonic acid being 16, oxygen 1·5, and nitrogen 82·5, the lowest carbonic acid 6·0, oxygen 5·5, nitrogen 88·5. Dr. Duncan⁶² estimated the foetid gas from a case of pneumothorax secondary to empyema to contain 26 parts of sulphuretted hydrogen and carbonic acid, and 74 of nitrogen.

In discussing the symptoms, prognosis and treatment of pneumothorax, it must be borne in mind that the conditions present are importantly varied according to the nature of the opening, whether

- a.* Direct and patent, or
- b.* Oblique and valvular.

(a). It is obvious that if the opening be direct and patent there can be no positive air-pressure within the pleura since no air can be pent up there. By means of a trochar fitted by tubing to a water-pressure gauge, I have ascertained *post-mortem* the degree of intra-pleural pressure present in 16 cases of pneumothorax. In 4 out of these cases the pressure was *nil*. On the other hand, (*b.*) if the opening be oblique or valvular, it is clear that although during inspiration air may enter the pleura, yet the moment the air is compressed in expiration the sides of the oblique opening or the tongue of the valve close and the air cannot escape at all or only with more or less difficulty according to the perfectness of the valve. In 12 cases out of the 16 above mentioned, there was more or less intra-pleural pressure present varying in degree from $1\frac{3}{4}$ inch to 7 inches of water.⁶³

The *first* effect of perforation of the pleura is effusion of air and, perhaps, the escape of some purulent fluid from the ruptured lung into the pleural cavity, the *second* effect is more or less intense pleuritis, and *thirdly*, if the patient survive, we usually get more or less purulent effusion.

⁶² *Edin. Med. and Sur. Jour.*, Vol. 28.—1827.

⁶³ *Medico-Chir. Trans.*, 1876, p. 179.

The principal *symptoms* of pneumothorax are—sudden acute pain in the side, followed, or rather attended by great dyspnœa and shock. The pulse becomes frequent, feeble, small, the respirations relatively more frequent than the pulse. The voice feeble or suppressed. There is occasionally great hyperæsthesia of the affected side. The position of the patient is frequently changed, he may sit up or recline, with the head raised and an inclination to the sound side, (after fluid effusion to the affected side): the sitting posture with slight inclination forwards and with the elbows resting upon the knees, is the most commonly chosen. There is nothing absolutely characteristic about these symptoms save, perhaps, the suddenness with which they may supervene. The patient is sometimes conscious of “something having given way,” and feels a peculiar trickling, cold sensation, associated with the pain in the affected side. All the symptoms of pneumothorax may be most closely simulated in an attack of acute pulmonary congestion supervening upon already advanced disease. On the other hand, there may be an almost entire absence of any special symptoms to mark the onset of the attack. In cases in which the pneumothorax supervenes in an advanced stage of the disease “palpitation” may be the chief complaint of the patient.

The *physical signs*, however, of pneumothorax are very positive, and can rarely be mistaken for those of any other disease. The alarmed, anxious and distressed countenance, the evident urgency of the dyspnœa usually amounting to orthopnœa, and the small, whispering voice are in themselves in marked cases, strikingly characteristic. I will enumerate the chief physical signs, and will dwell only upon those which are most essentially important.—The affected side is enlarged, the shoulder raised, the intercostals effaced, and little or no movement is perceptible with respiration, whilst the opposite side labours with the rapid breathing, the soft parts receding with each inspiration. The heart is displaced towards the sound side, and the abdominal organs are depressed. The

percussion note is greatly hyperresonant or truly tympanitic, where it had perhaps before been dull. Respiratory murmur is absent, or very feeble, or amphoric breathing is heard at one or two points, with metallic whisper and metallic echo on coughing. Pectoriloquy is scarcely ever present. Metallic tinkling may be heard, especially after cough. Vocal fremitus is either absent or much diminished.

Later, when effusion of fluid has occurred, there is dulness at the lower, hyperresonance at the upper, part on the affected side, the distribution of dulness and resonance shifting with the altered position of the patient. On placing the ear down upon the chest and giving the patient a somewhat abrupt shake, a splashing sound may be distinctly heard, perfectly characteristic of hydro-pneumothorax. This "succussion" sign may be observed in cases in which we can find no other evidence of the presence of fluid in the pleura. The explanation of this is simple enough: the moment pneumothorax occurs, the diaphragm on that side becomes flaccid and more or less concave, drawn downwards by the weight of the abdominal organs, and in its concavity a not inconsiderable quantity of fluid may collect not to be detected by percussion dulness. In cases in which there is a considerable quantity of fluid present intercostal fluctuation may be made out, or, on sharply percussing immediately below the line of contact of resonance and dulness, a thrill may sometimes be detected by the finger significant of fluid vibrations.

Hyper-resonance, absent, feeble, or amphoric respiration sounds, and *displacement of heart* are, however, the signs of pneumothorax of central importance about which the other signs of less value may be grouped. These three signs alone are sufficient to render the diagnosis certain, and their presence can be ascertained by a physical examination which will not add to the distress of the patient.

The degree and extent of the *hyper-resonance* depend upon the quantity and tension of the air that has escaped into the

pleural cavity. In some cases adhesions are so strong and extensive as to limit the pneumothorax to a small portion only, usually the base, of the pleural cavity. In such cases the symptoms are rarely urgent, and their cause may escape notice. But as a rule the whole lung is collapsed save, perhaps, at the summit where there are frequently some old adhesions. The hyper-resonance has a drum-like quality which is usually characteristic, but in cases in which the tension of the air is very great the vibrations of the chest-wall are less free and the tympanitic note somewhat deadened. The boundaries of hyper-resonance include, and may extend beyond the sternum towards the healthy side, and if the left side be affected the normal cardiac dulness is altogether effaced.

The character of the *breath-sounds* varies according to the nature of the opening; over the greater portion of the affected side the respiratory sounds are as a rule annulled, and in those cases in which the opening is small and quite valvular no auscultatory sounds may be detected at any point, although very often even in these cases, at one spot a faint and distant hollow inspiratory sound may be detected on careful auscultation. In cases, however, in which the opening through the pleura is free, the entry and exit of the air to and from the pleural cavity gives rise to a variety of amphoric breathing peculiarly large and of metallic quality which can rarely be mistaken. This amphoric breathing is most audible at some one portion of the chest nearest to the seat of perforation, it is commonly best heard at the mammary, or upper or lower scapular, region, and is conducted more or less distinctly to a distance from this point. The expiratory portion of the amphoric sound is peculiarly indicative of a free and patent opening. In these latter cases, with free opening, the voice sounds may be attended with a metallic echo quite peculiar, whereas in the more valvular cases the voice sounds are not conducted at all. Metallic tinkling may sometimes be present and is a useful additional sign; one, however, which I have heard very typically

yielded by large pulmonary cavities. Feeble, and more or less modified, breath-sounds may be heard at the apex where there are still adhesions, and, immediately after the occurrence of perforation, friction sounds may sometimes be heard over portions of lung as yet in contact with the thoracic wall.

Displacement of the Heart.—M. Gaide⁶⁴ was the first to describe displacement of the heart as an important sign of pneumothorax, and he relates an interesting case in which the patient, a young woman, at the moment of the occurrence of pneumothorax, was conscious of the heart's beat having been transferred to the right of the sternum. M. Rayer in whose clinique the case occurred, was disposed, however, to regard the displacement, new to his experience, as congenital, but *post-mortem* inspection proved it to have been the result of the escape of air into the left pleura. M. Gaide relates another case of left sided pneumothorax in which this sign was observed. It is indeed a constant and, save in exceptional cases in which the base of the opposite lung is consolidated, an essential sign of perforation of the pleura, and it is singular that it should have escaped the notice of such acute clinical observers as Laennec and Louis. Its occurrence simultaneously with that of the perforation, already noticed but not explained by M. Gaide, is a fact that would of itself cast suspicion upon the usual acceptance of the sign as being necessarily one of pressure. The cardiac displacement is due in the first instance, to the sudden removal from the mediastinum of the elastic traction of the lung which has collapsed, and the consequent unopposed traction upon it of the other lung. And, if the opposite lung be not solidified, the heart may from this cause alone be carried beyond the median line. Thus, I have recorded three cases in which the heart was displaced to the right of the sternum, yet in which as proved by experiment *post-mortem* no intra thoracic pressure existed.

I may here perhaps be allowed to quote one of these cases

⁶⁴ "Obs. à l'Hôpital St. Antoine." Archives gén. de Medicine, t. xvii. 1828.

recorded in the *Medical Times and Gazette* for May 21st, 1869—to illustrate what I conceive to be a clinical fact, viz., that there may be great lateral displacement of heart in pneumothorax quite independently of intra-thoracic pressure.

Case 1.—Walter C., aged 21, admitted into the Brompton Hospital, under the care of Dr. Cotton, February 12th, 1869. Patient had a strong hereditary predisposition to phthisis and dated his present illness from thirteen months previous to admission. On January 10th while at the Chelsea Home, awaiting his turn for admission, he was seized with pain in the left side. He was seen on Jan. 12 by Mr. Charles Joubert, then resident Clinical Assistant at the Hospital, who found the whole left front of the chest tympanitic on percussion, with distinct amphoric respiration, most plainly audible below the clavicle. The hyperresonance did not extend beyond the mid-axillary vertical line; there was dulness posteriorly with scattered humid crepitation. The heart's impulse was seen, and felt at the fifth right intercostal space within the nipple; the patient had remarked at the time of his seizure that the heart beat to the right of the sternum. Decubitus right; respirations thirty in the minute; pulse 100. There were signs of excavation and softening at the right apex.

Jan. 15. Better; no urgent symptoms of pneumothorax.

On admission into the Hospital (February 12th), the hyperresonance on the left side extended beyond the median line in front, the amphoric respiration was well marked in front and laterally to the base, and metallic echo and tinkle were audible. The deficient resonance and moist râles were still present posteriorly. The disease in the opposite lung had advanced. The heart's maximum impulse was to the right of the sternum; the apex was however, ascertained by percussion and palpation to be at the ensiform cartilage. The respirations were 30 in the minute, the pulse 96. No urgent dyspnoea. The patient was weaker and lingered on, gradually sinking from the progress of the general disease, without any ma-

terial change in the physical signs. He died May 26th, 1869.

Autopsy thirty hours after death—No difference noticeable in the relative size of the two sides of the chest. A trocar and canula connected by tubing with a water-pressure-gauge, was inserted at the fifth left interspace, to ascertain the air-pressure within the pleura. This was found to be *nil*. A stilette was then thrust in at the fourth right interspace near the sternum, the trocar withdrawn, and the cartilages removed in the ordinary way, the heart being transfixated in position by the stilette. The exact position of the heart was as follows: *Vide* Fig. 6. The apex was behind the sternum, and slightly to the left of the median line—*i.e.*, in the vertical line of the left sternoclavicular articulation, and at the level of the fifth rib. The left border of the heart occupied the median line, with a slight inclination to the left; the right border was touched by a line drawn vertically from the middle of the right clavicle. The left pleura contained a small quantity of purulent fluid; the lung was collapsed backwards, and a large opening capable of admitting the end of the little finger was seen near the apex, through which air bubbled freely on blowing into the trachea. The right lung was excavated at the apex, it was partially collapsed below, and presented scattered patches of grey tubercle and pneumonia.

FIG. 5.

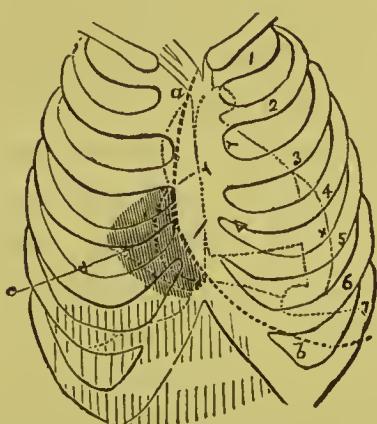
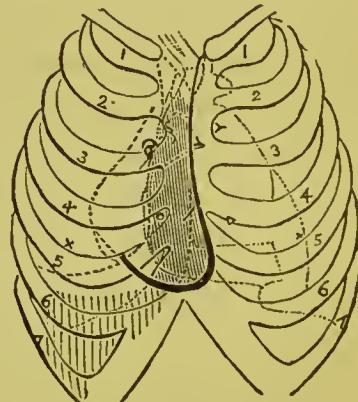


Diagram representing percussion limits marked out before opening thorax. *a, b*, Boundary of left pleura. *c*, Displaced cardiac dulness.

FIG. 6.



Showing exact position of heart on removing sternum. A small puncture is indicated at fourth space through which a stilette was thrust to fix the heart *in situ* before removing sternum.

I am aware that Dr. Hayden in his able and elaborate work on Diseases of the Heart, p. 102, dissents from the views I have elsewhere expressed⁶⁵ respecting the mechanism of cardiac displacement in pneumothorax. I shall consider Dr. Hayden's objections more fully in the volume on heart diseases, but I may observe that at page 7 of the above mentioned work, he appears, as will be seen by the following paragraph, to acquiesce in the presence of those physiological conditions by a disturbance of which I contend that the cardiac dislocation is effected. Dr. Hayden says "Inferiorly the pericardium is fixed and held tense by its attachment to the great phrenic centre of the diaphragm; and by the tension of the pleura reflected from it upon the lung on either side, it is subjected to constant, considerable, and in the normal state, equal traction laterally."— It seems to follow as a consequence so necessary as to need no further demonstration, that if the traction on one side be annulled by the admission of air into the pleura, the balance is deranged and the heart pulled over by the traction from the opposite side. I say it is the lung traction from the healthy side that, thus set free, draws over the heart; Dr. Hayden is of opinion that it is atmospheric pressure unopposed by lung elasticity on the diseased side that pushes the heart across the median line.

I need scarcely point out the great importance of recognizing the fact that dislocation of heart does not necessarily mean positive pressure upon that organ by air pent up in the pleura. Were it otherwise, paracentesis would be indicated in every case of pneumothorax, whereas we know that the operation is valuable in but a small proportion of cases.

In those cases in which, on account of the valvular nature of the perforation, the air which enters the pleura with inspiration, cannot again escape with expiration, or can only do so partially and with difficulty, a gradually increasing accumulation of air takes place and gives rise to very considerable

⁶⁵ *Med. Times and Gazette and Brit. Med. Journal*, 1869.

direct pressure upon the heart. This pressure never, I believe, equals that which is sometimes present in fluid effusions, the highest I have measured having been equal to seven inches of water.

Displacement of heart is not always easy of detection in right sided cases of pneumothorax, but a little care will usually enable one to detect the apex beating in the left axillary line, whilst the right margin of normal percussion dulness is equally displaced towards the left. In one very rare case of left sided disease, there was associated with the perforation a communication (probably congenital) between the pericardium, and the pleura, through which the air penetrated and, pressing back the heart, completely obscured both dulness and impulse, and rendered the sounds barely audible.⁶⁶

The intensity of the symptoms in pneumothorax is dependent upon:—(a.) The greater or less amount of respiratory area suddenly cut off. (b.) The degree of pressure upon the heart and resistance to the flow of blood through the capillaries of the affected lung.

It is obvious that the greater the amount of healthy lung suddenly collapsed, the greater the degree of dyspnoea and shock. The pressure upon the heart is a manifest cause of distress, it being precisely the reverse of what normally obtains, namely an aspiration, from the elastic traction of the lungs on each side, of the blood from the great veins towards the cavities of the heart. But whilst, as has been shown by the experiments of Goodwyn⁶⁷ and Erichsen,⁶⁸ the mere collapse of a lung affects but little the facility of the circulation through it, its compression or forcible collapse necessarily retards the circulation, and throws extra work upon the already troubled heart.

In August 1867, I performed some experiments bearing

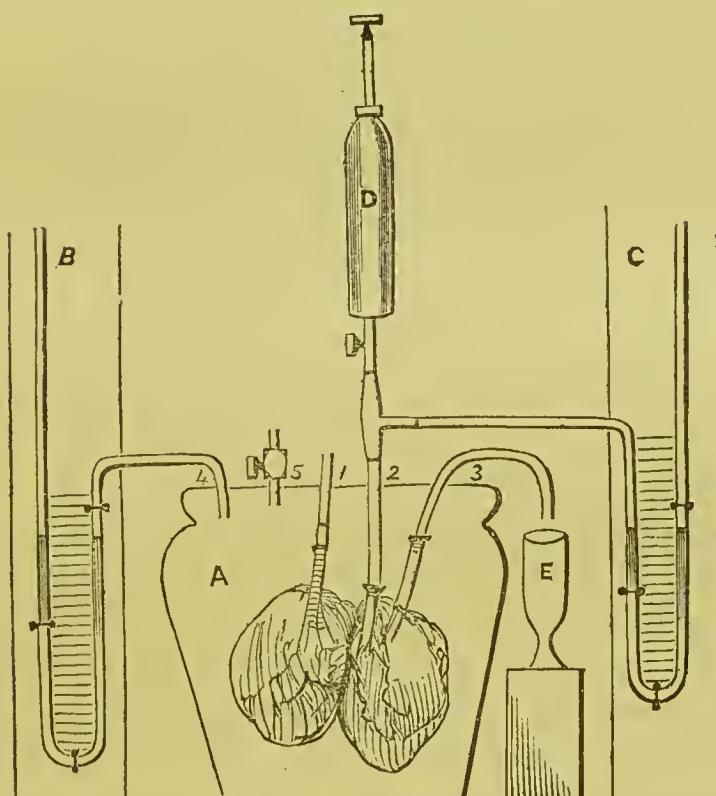
⁶⁶ *Path. Trans.*, Vol. xx., p. 99.

⁶⁷ *The connection of Life with Respiration.* 1788.

⁶⁸ *Experimental Enquiry into the pathology and treatment of asphyxia.*

upon this point. A sheep's lung was placed in a wide-mouthed glass jar closed by a piece of bladder through apertures in which tubes 1, 2, 3, (Fig. 7), were inserted and connected with the trachea, pulmonary artery and pulmonary vein respectively. To the trachea-tube the pipe of a syringe was applied, having a lateral branch connected with a pressure guage.

FIG. 7.



The tube attached to the pulmonary vein opened at its other end over a receiver E. Two other tubes communicated with the interior of the jar, one (4) being connected with a second dynamometer B, the other (5) being supplied with a stopcock.

By means of the syringe, whipped blood was injected steadily and at a uniform pressure of $1\frac{3}{4}$ inches of mercury⁶⁹ (noted by dynamometer C), into the pulmonary artery, and the time required by the fluid escaping through the pulmonary vein

⁶⁹ This pressure was chosen as representing pretty nearly the power of the right ventricle as observed by Colin—*Comptes Rendus*, T. lix. 1864.

to fill the receiver E was noted,—(*a*), with the lung moderately expanded, to be 88 seconds: (*b*), with the lung collapsed by its own elasticity, 85 seconds: (*c*), with the lung compressed by air injected through tube g into the jar until dynamometer B registered a pressure of 9 inches of water, 230 seconds: (*d*), with the lung similarly compressed by 5 inches of water, 190 seconds:—lastly, (*e*), the trachea tube was secured by a ligature, and the air pressure then removed from the surface of the lung by opening the stopcock, g . when the time required to fill the measure E was observed to be 125 seconds.

These experiments in which, as in two other series conducted at higher pressures I was aided by my friend the late Mr. Alexander Bruce, shew that whereas the difference in resistance to circulation through a moderately expanded and a *simply* collapsed lung was scarcely perceptible, yet that a moderate pressure upon the lung of five inches of water quite doubled this resistance. That the increased difficulty of circulation was largely due to the more thorough collapse of the lung, rather than to its compression, seemed proved by its persistence, (Exp. *e*), after the direct pressure had been removed from the lung. I have shewn that the intra-thoracic pressure in pneumothorax may vary from *nil* to 2, 4, 5, or 7 inches of water, and in some cases it may probably be found to mount even higher than this. It is clear then that a corresponding pressure must be thrown upon the right ventricle and upon the capillaries of the opposite lung, this disturbing influence being, however, less manifest in those cases in which the perforated lung had already become much diseased and its vessels largely occluded.

The state of the opposite lung largely influences the urgency of the symptoms. It not infrequently happens that the pneumothorax occurs on the comparatively sound side, and in such cases, happily death speedily brings to an end the hopeless and agonising distress of the patient.

Progress.—If the patient survive the dyspnœa and shock of the first attack—and he usually does so, provided the opposite lung is not extensively diseased—after the lapse of some

thirty six hours or two days, signs of reaction, fever and hectic, announce the supervention of acute pleuritis and commencing empyema. The cause of the suppurative pleuritis which generally follows upon perforation of the lung is, in part the irritation of the air admitted into the pleura, in part also the tearing of adhesions, but the most potent cause is the escape of some of the contents of a cavity into the pleural sac. The signs of fluid in the pleura soon become apparent, and in some cases the pleura becomes rapidly filled with fluid, what air remains being compressed into such a small compass at the summit of the thorax as to be very difficult to detect. A large portion of the air in these cases is either absorbed, or as suggested by Dr. Duncan⁷⁰ it may be expressed through the opening in the pleura. Dr. Duncan doubts whether the diseased pleura in pneumothorax can absorb air, but it must be remembered that such absorption is encouraged by the gradual pressure of accumulating fluid. Moreover, the aperture is frequently much too low down to permit of expression of any large portion of the air through it. One effect also of the pleurisy is, sometimes, to completely close the original perforation. The case may thus in the course of time be completely transformed from one of pneumo- to one of pyo-thorax. In other instances very little effusion takes place and the signs of pneumothorax remain marked to the end.

Diagnosis.—There can be but little difficulty in distinguishing a case of pneumothorax in which the effusion of air is extensive from any other disease. *Emphysema* is the only other disease in which we get hyper-resonance* and enfeebled breathing combined, but emphysema is a disease which affects both lungs, the respiration is never quite suppressed nor of amphoric quality, nor is the heart ever displaced as in pneumothorax. The diagnosis is sometimes difficult, in-

⁷⁰ *Edin. Med. and Surg. Journ.* Vol. 28—1827.

* The resonance of emphysema also lacks the drum-like note found in pneumothorax.

deed impossible, between localised pneumothorax and a large thin-walled pulmonary *cavity*. Such cavities may yield almost tympanitic resonance, and very typical metallic tinkling rhonchus. Localised pneumothorax is most commonly situated, however, at the lower portion of the thorax, and in this situation such a large cavity as could be confounded with pneumothorax is of most rare occurrence, unless it be continuous from the apex downwards in which case the heart's beat would be over an extended area on the *affected* side. The cases which I have oftenest seen mistaken for pneumothorax, however, have been cases of advanced phthisis, in which *acute congestion* has rapidly supervened at the base of the comparatively sound lung. Pain limits the movements and lessens the sounds over the newly affected part, there is considerable high-pitched resonance on percussion, and the symptoms may be precisely those of pneumothorax. Breath-sound and rhonchus can be heard, however, on careful auscultation, the heart is not displaced, nor is the percussion note truly tympanitic. Sometimes at first sight the dyspnœa of *asthma* nearly resembles that of pneumothorax, and, with general hyper-resonance, we may have in asthma an absence of respiration over portions of the chest; but the portions of lung so affected will vary in position, perhaps, even while we are listening, and the general wheezing râles present elsewhere, together with the history of the case and the effect of treatment, will prevent any real difficulty in diagnosis. I have seen more than one case of *hysterical dyspnœa* closely simulating pneumothorax, but the expression of countenance cannot be simulated, and a moderately careful physical examination will lead to a right diagnosis. The diagnosis of hydro- or rather of pyo-pneumothorax from simple empyema is not difficult, the succussion splash, and the marked shifting of the dulness and resonance with change of position, being quite characteristic of the former disease. But, as already observed, some cases of apparently pure empyema have their origin in perforation of the lung.

The diagnosis in pneumothorax, however, does not consist merely in separating it from other diseases, but also in distinguishing the kind of perforation that has taken place, and the probable existence or not of air-pressure within the thorax. The discovery of amphoric (to and fro) breathing renders it pretty certain that the opening is a free one, admitting the ready passage of air both ways, and that consequently no air-pressure is present. The complete absence of all breath-sound, with increasingly urgent dyspnœa, distended side and greatly displaced and oppressed heart, are equally significant of a valvular opening and of increasing intra-thoracic pressure. In a third set of cases hollow inspiration and obstructed rhonchoid expiration suggest a partially valvular opening.

Prognosis.—Although as a rule pneumothorax is an occurrence of speedily fatal augury, yet in some cases life is very considerably prolonged, the perforation and its consequences proving to be lesions of a conservative character. Cases in which a valvular opening is detected, are by no means necessarily the worst for prognosis, provided that they are promptly treated. The condition of the other lung is the point to ascertain in calculating the future progress of the case; if tolerably sound, one may hope for arrest of the pulmonary disease and conversion of the case into one of chronic empyema.

A glance at the table at the end of this chapter shews, however, that in the majority of instances, pneumo-thorax occurs towards the close of the disease, when the patient is already dying from extensive pulmonary lesions. In a few instances the pneumo-thorax does not seem to have shortened life (cases 12 and 31). In cases 14 and 36, a large fluid effusion succeeded to the pneumo-thorax, in each instance converting the case into one of empyema. A similar tendency to the accumulation of fluid may be observed in the majority of the cases, and I am inclined to think that not a few of the cases of empyema that come before us in which after paracentesis we

find evidence of apex disease, the empyema has originated in pneumo-thorax; the effect of the pneumo-thorax and subsequent effusion having been to arrest the pulmonary disease. I have at all events met with two or three cases that would be best explained in this way:—In one instance, the patient left the Hospital “cured” after the removal of 6 pints of a sero-purulent effusion by paracentesis. He had presented, however, signs of early (or old?) disease at the apex of the lung soon after the tapping, and although he resumed his work for a time after leaving the Hospital, he again came under observation 12 months later with pneumo-thorax on that side, and died in three weeks.

Treatment.—Life is threatened on the occurrence of pneumo-thorax by *shock*, *asphyxia* and *exhaustion*, and these are special indications for treatment. The shock—which is due to the sudden lesion of a vital organ, and to the sudden dislocation and impediment to the action of the heart—must be treated by the administration of a stimulant, but above all things by an opiate. Opium is most valuable in calming the nervous system and in lessening the sense of dyspnœa. It may be given in the form of pill with camphor, or in divided doses of chlorodyne, or a dose of morphia may be administered subcutaneously. When the immediate shock has been relieved, the patient must be carefully examined and watched for signs of increasing pressure within the chest, to be treated by the timely introduction of a fine trochar. This trifling operation gives great, and, curiously, sometimes lasting, relief. It can be repeated, however, if necessary, or a fine trochar guarded with gold beater’s skin may be left in the side. If we bear in mind that the chief way in which positive pressure is brought about within the pleura, is by the thoracic wall on the affected side being expanded to the position of extreme inspiration and then recoiling upon the air pent up in the pleura, the advantage, in these cases when the excess of air has been removed of strapping the affected side so as to control inspiratory movement, becomes obvious. A broad band of strapping firmly

applied over the lower ribs, and reaching some two or three inches beyond the median line in front and behind, is sufficient to restrain the movement of the affected side.

As a rule pneumothorax occurs in persons already reduced in flesh and blood by previous illness ; if the accident should occur at an earlier period of the disease, the venous engorgement, lividity, and general circulatory distress will be correspondingly marked. In such cases free dry cupping will give great relief. The portal system is the great reservoir for retarded blood, and an occasional saline aperient is of value in pneumothorax, serving also to correct the constipating effects of opium, the administration of which latter drug in repeated small doses, is on other grounds desirable for the first few days.

The intensity of the secondary pleurisy must be moderated by fomentations or poultices, and the associated fever, pain, and hectic, treated by occasional doses of quinine and opium, with salines or mineral acid as the case may require. The occurrence of effusion may be regarded rather with satisfaction than otherwise, provided the opposite lung be tolerably sound ; some cases of one sided phthisis have undoubtedly been arrested by the occurrence of pneumothorax and consequent empyema, the compression of the lung checking all active disease. Hence we should not be too hasty to remove the fluid by paracentesis since, notwithstanding the presence of air, it has little tendency to become offensive, and the dyspnoea is lessened and the perforation more likely to become closed, by the substitution of steady for elastic compression of the lung. Paracentesis performed too soon, or with too great an anxiety to remove all the fluid, will lead to a recurrence of the pneumothorax by reopening the old perforation. At a later period paracentesis may be necessary.

The frequent administration of food in small quantities with sufficient stimulant to steady but not to excite the heart's action, is needed to prevent increased suffering from exhaustion.

TABLE OF CASES OF PNEUMOTHORAX.

No.	Sex.	Age.	Duration of illness.	Stage of disease.	Side most diseased.	Lung perforated.	Duration.	Remarks.	
1	F.	13	4 years	Cavities, both lungs	Both equally	Left	10 days	No fluid effusion	
2	M.	20	19 months	{ Cavities, left; emphysema " and tubercle, right }	Both	"	8 hours	{ Perforation small, diaphragm, convex towards abdomen 1½ pints of pus in left pleura	
3	M.	23	8	Double cavities	Left	"	14 days	2 pints fetid pus in left pleura	
4	F.	25	5	{ Admitted in a dying state with double cavities }	"	Right	3 weeks		
5	M.	20	10	Advanced cavities, both lungs	Both	Left	2 days		
6	M.	21	?	Cavities, both	Left	"	2 weeks		
7	F.	22	18 months	{ Cavities, left; right only }	Right	"	25 days	10 oz. pus in pleura	
8	M.	22	6	{ slightly diseased }	Left	Left	10 "	Pleura filled with sero-pus	
9	M.	22	3 years	Double vomica	Both	Right	2 "	2 pints of serum in pleura	
10	M.	35	6 months	{ Rapidly forming cavity at right apex }	Right	"	6 hours	1 pint fluid in pleura	
11	F.	29		(Winter cough many years).				Opening valvular	
12	F.	20	12	{ Softening right lung at base. Tubercular meningitis, and finally pneumonia right }	"	Left	43 days	12 oz. of pus in pleura	
13	M.	44	14	Double vomica	Both	Right	?	6 pints purulent fluid in pleura	
14	M.	26	6	Cavity, both sides	Right	"	4 months	Opening not found	
15	M.	23	7	Cavities, both lungs	Both	Left	15 minutes	No fluid in pleura	
16	F.	13	8	{ Cavity, right lung; tubercle }	?	Left	3 days	22 oz. of purulent fluid in pleura	
17	F.	?	12	{ Left }	Right	Left	14 "	40 oz. pus in pleura	
18	M.	21	4	{ Moribund on admission with pneumothorax }	"	"	3 weeks	12 oz. of turbid serum in pleura	
19	M.	22	6	Cavities, both lungs	Both	Left	1 hour	1 oz. of pus in pleural cavity	
20	F.	37	13	Double cavity	Right	4 days	4 days	2 parts pus in pleural cavity	
21	M.	41	6	"	Left	8 "	8 "	4 parts pus in pleural cavity	
22	M.	35	4	Cavity left, softening right	Left	Left	?	4 parts pus in pleural cavity	

1 pint purulent fluid in pleura
no return of active
actions

1 pint
2 pints

26	M.	17	9	,	Double vomica	Left	Left	?
27	M.	18	7	,	„ „	Right	„	?
28	M.	25	II	,	Double small vomica	Left	Right	21 days
29	M.	22	8	,	Consolidated right apex Softening both apices, cavity left	Right	„	2 „
30	M.	44	15 months		Cavities at apex of right, pneumothorax (one month) left	Left	Left	2 „
31	M.	21	13	,	Admitted with left pneumo- thorax	„	„	Case referred to at page 131
32	F.	24	8	,	Double cavities, extensive disease both	Both	Left old, right 10 minutes	4½ months
33	M.	34	2 or 3 years		Both lungs much diseased	Right	36 hours	Opening valvular
34	M.	17	?		Both lungs both apices	Left	3 days	Communication between peri- cardium and pleura on left side.
35	F.	19	6 months		Cavity left; right nearly free below on left side	„	14 „	1 pint fluid in pleura
36	F.	17	9	,	Cavities both and softening both apices	„	„	5½ pints of pus in pleura, open- ing closed, death from em- pyema
37	M.	23	8	,	Softening both apices	Right	12 months	Only portion of healthy lung remaining, collapsed by pneu- mothorax
38	F.	25	Some months		Both lungs slightly consoli- dated	Left	30 hours	
39	M.	25	3 years			„	Few days	
								1½ pints serous fluid in left pleura

SUMMARY OF TABLE.

39 cases—(Collected from the *post-mortem* records of the Brompton Hospital) 27 males, 12 females; 22 right side, 15 left side, and 1 double-sided. Greatest duration of life after pneumothorax, 12 months, 4½ months and 4 months; least duration, 10 minutes, 15 minutes, 6 hours; mean duration about 27 days. In two instances no fluid effusion in the pleura, duration 10 and 3 days respectively. In three instances, specially noted, that pleura contained serum, duration 2, 10, and (?) days respectively.

Note.—Dr. H. Bernheim of Nancy refers to 36 cases tabulated by De Saussier, of which 3 died in a few hours, 38 within ten days, 19 lived from one to five months, 2 from eight to eleven months; and I lived two years. According to Louis the duration of life varies from sixteen hours to thirty-six days only one of his cases having survived the latter period.—*Leçons de Clinique Médicale*, p. 131.

CHAPTER XII.

ULCERATION OF THE BOWEL IN PHTHISIS.

Ulceration of the bowel in phthisis. Pathology unsettled. Morbid anatomy more definite. Appearances described. Seats of ulceration. Etiology. Symptoms vary somewhat with locality of ulceration. Rules for diagnosis. Chest signs and symptoms abate during diarrhoea, an important feature in diagnosis. Extensive ulceration may exist without diarrhoea. Caution needed in employment of purgatives in phthisis. Treatment. Mild cases. More severe cases. Importance of rest.

ULCERATION of the bowel is a very common complication of phthisis, indeed it may be said to be one of the attendant lesions of the disease, occurring during some period in the course of a large proportion of cases. Out of one hundred *post-mortem* examinations of which I have careful notes, ulceration of the intestine was present in forty.⁷¹

The pathology of the disease is involved in the same difficulty and doubt as that of tubercular laryngitis, opinions being quite as opposed as to whether in its origin it is primarily tubercular or not. In regard to the broader anatomical facts of this lesion, however, there is not much room for difference of view.

The disease commences with inflammatory swelling of the follicles of the small or great intestine, causing them to enlarge and to become choked with their proliferated parenchyma, caseation follows, and the softened products discharge

⁷¹ It might have been present in a few more instances, as I have placed a query opposite to some of my negative cases, shewing that the intestines have not been sufficiently examined.

into the intestine, leaving an ulcerous recess behind. The margins and base of this primary ulcer consist "of corpuscular infiltration of the connective tissue, but neither in the corpuscular elements themselves, nor in the mode of their arrangement, can we detect anything specific" (Kindfleisch).⁷² Other observers, however, consider this "corpuscular infiltration" as truly tubercular, and others again consider the whole process as being, from the first, of this specific nature. Beyond this point, however, authorities are less at variance. On the peritoneal surface of the intestine, over the site of an ulcer thus established, some flakes of lymph are to be seen, or inflammatory adhesions may be contracted with an adjacent coil of intestine. Beneath the peritoneal surface, and shining through it, the outline of the ulcer can be seen, and in and about its base granulations of tubercle are to be observed. These granulations are connected with the lymphatic vessels, some of which form white lines or streaks over and about the site of the ulcer. The ulceration tends to extend in a transverse direction, beyond the limits of the gland follicle in which it originated. This extension follows the direction of the vessels, and is determined by the formation of tubercles in their sheaths.

The question is, whether these out-springing tubercles are but an extension of a disease from the first tubercular, or whether, as we have held to be the case in the lungs, a catarrhal inflammation brings about the first follicular destruction, to which the tuberculosis is secondary,—an infective process starting in the lymphatic tissues, and giving to the original lesion its inveterateness and disposition to spread? The microscope has as yet failed with sufficient definiteness to solve these doubts, but tends to favour the latter view.

Whether the ulcers shall be single or in groups depends upon the glands, whether single or agminated, which are

⁷² *Pathological Histology*, Tuberculosis of Mucous Membrane, p. 447, Sydenham Society's Edit.

attacked. Peyer's patches are favourite seats of ulceration. In them, irregular serpentine ulceration arises by extension and coalescence from several centres, often leaving small tracts or islets of mucous membrane intact. All the gland follicles of a patch are by no means necessarily affected, the ulceration may only involve a certain number of them extending transversely to the mucous membrane beyond. In the typhoid ulcer on the contrary, the whole patch is involved simultaneously and uniformly, and the ulceration is limited to the patch. In the cœcum too, the ulceration is often very extensive, the whole of the mucous membrane being eroded for several inches with the exception of small islets or streaks here and there which have escaped, marking the original extension of the ulcerative process from many centres. As already mentioned local peritonitis commonly attends the intestinal lesion, and adhesions are frequently formed. Perforation of the bowel is more common than is generally thought, sometimes leading to the escape of fæcal matter and to general peritonitis, sometimes to the establishment of a fistulous communication between adherent portions of intestine, and again in other cases producing collections of pus localised by surrounding adhesions. These processes are strictly analogous with those which occur in connection with the pleural surface of the lungs in phthisis.

The lower two or three feet of the ileum and the cœcum *coli* are almost invariably the portions of bowel involved, but the ulceration may extend both beyond and above these points to the rectum and the duodenum. The tendency as a rule, is however, to extension downwards, so far at least as the term extension is applicable to this lesion. For in the ileum for instance, the mucous membrane between Peyer's patches is commonly intact. My own observation would lead me to regard the *cœcum coli* as the favourite seat of this lesion, it is there that tubercular ulceration will be often found when it exists nowhere else in the intestines, and it is there

that the lesions, when also present at other parts, are commonly found to be most advanced, and therefore presumably of oldest date.

More or less general hyperæmia of the mucous membrane attends the ulcerative process, very variable in amount at different times, and in different cases. Considerable general thickening, especially of the large intestines, is sometimes present.

Ulceration of the intestines of the kind above described, is scarcely ever met with in adults and only very rarely in children, save in association with pulmonary disease, it most usually complicates the later stages of the disease; but it may, like laryngitis, occur at a very early period at a time when the pulmonary physical signs—always somewhat masked during diarrhœa, are difficult to detect. Anything that tends to derange the digestion, and the bowels, favours and may determine the occurrence of the disease. Klebs⁷³ and Mosler assert that swallowing the expectoration from phthisical lungs may lead to intestinal lesions, and I am decidedly of the same opinion although proofs are wanting. The diarrhœa of phthisis is more prevalent in the late summer and autumn months than in the winter.⁷⁴

The usual symptoms of ulceration of the intestines are *diarrhœa* and *pain in the abdomen*.

There is nothing at first which can be noted as peculiar in the character of the diarrhœa, pale loose stools, resembling those of ordinary intestinal catarrh and being dependent doubtless upon the presence of such catarrh. There may be some nausea; the tongue is furred with red tip and edges and prominent papillæ, and the patient complains of thirst. The pain complained of is usually referred to the umbilical region, it is of a colicky nature, but there is no

⁷³ Quoted in Jones' and Sieveking's *Pathological Anatomy*, 2nd edit., edited by Dr. Payne.

⁷⁴ *Berliner Klinische Wochenschrift, loc. cit.*, Oct. 1873.

marked tenderness over any spot of the abdomen. There is marked irritability of the whole mucous tract, the taking of food, and more often warm drinks, into the stomach causes the bowels soon after to act. The looseness is, however, at this stage tolerably amenable to treatment, and for a time the motions become natural, or the patient is even constipated. Soon, however, a relapse takes place, and the diarrhoea is more obstinate than before. Now some decided tenderness may be felt on deep palpation, most likely in the right iliac region, the motions become more scanty, some mucous is passed with them, a speck or two of blood may be observed, or a teaspoonful or more may escape. Anything approaching to copious haemorrhage is, however, very rare. The tongue becomes red and patchy from loss of epithelium, it may present short transverse fissures on each side of the median line. The further symptoms vary with the seat of the principal ulceration. If this be limited to the ileum, the diarrhoea may for a long time be held in check by treatment, but the bowels are irritable, the abdomen somewhat prominent in the umbilical region and tender. If the ulceration, as is commonly the case, have its principal seat in the cœcum, the tenderness over that region is more marked, the diarrhoea is very difficult to control, blood and mucous are frequently present in the stools, and the pinched look of the patient and his rapid emaciation are more marked. Ulceration extending further through the colon to the rectum is signified by more distinctly dysenteric symptoms, pain and tenderness over the arch of the colon, tenesmus, more frequent and mucous stools, whilst on the other hand, the gastric symptoms are less marked, and considerable appetite with a fairly clean tongue is often found.

It is easy to note down these symptoms as they occur in case after case of this dreaded complication, but it is much more difficult to fix upon any symptom if any indeed exist, that is positively characteristic of ulceration. The plain rule is of

course, given chest disease, always treat intercurrent diarrhoea as though it were due to commencing ulceration of the intestines. But in some cases, the diarrhoea precedes, or at the time of observation altogether masks the chest disease. It is remarkable how completely even decided pulmonary disease may be masked by diarrhoea. The cough and expectoration may cease or become trivial, and the dryness of the pulmonary tissue gives an exaggerated "vesicularity" to the respiratory murmur that masks existing defect. When once we are aware of this fact, however, it becomes of much importance in diagnosis, the alternation in prominence between the chest and abdominal symptoms is very characteristic of intestinal phthisis. So far as my limited experience of dysentery goes, I have also observed that with the appearance of chest symptoms—cough and expectoration—the dysenteric signs markedly abate. It is unnecessary to dwell upon the later stages of this disease, rapid wasting and exhaustion from the constant uncontrollable diarrhoea, a depression of the previously somewhat raised temperature, aphous mouth and lividity of extremities are the closing symptoms.

It is a fact, however, worthy of note that even extensive ulceration of the intestines may exist without any diarrhoea. Dr. Walshe observes "Not only may pretty extensive ulceration exist in the ileum without pain, either spontaneous or elicited by pressure, but with a confined state of bowels: Again, I have known, in a case running an acute course, marked abdominal pain and tenderness conjoined with obstinate constipation, when after death, the bowels, in spite too of the frequent use of purgatives, contained abundant solid faeces, and the ileum was extensively tuberculated and ulcerated."⁷⁵ I have myself frequently observed constipation to be associated with ulceration of the bowels, most commonly however alternating with attacks of more or less severe diarrhoea.

⁷⁵ *Diseases of the Lungs.* 3rd edit., p. 455.

In the *Pathological Transactions* for 1868⁷⁶ I have recorded a case of chronic phthisis with extensive and deep ulceration of the ileum in which constipation was a marked symptom throughout the patient's illness. There is no doubt that the diarrhoea is, to a large extent, dependent upon irritative catarrhal inflammation of the mucous membrane in the neighbourhood of the ulcers. In some cases, however, the ulcerations are so deep as extensively to destroy the muscular coat and thus materially to interfere with the peristaltic movements of the intestines. In other cases of a more acute kind, the peritoneum may be much involved, and the muscular coat thus paralysed. An analogous state of things in both these respects sometimes obtains in typhoid fever. It is not at all uncommon for a case of typhoid fever, well marked in all other features, to be attended with constipation throughout, and I have known such a case to terminate fatally by perforation.

These latter considerations bring home to us the great importance of a very careful treatment of constipation in phthisis, the stronger purgatives should only be administered with the greatest caution. I have seen perforation occur from the too hasty administration of a couple of the pil. coloc. c. hydrarg. for constipation, which was present together with ulceration. This symptom should be combated with castor oil, or small doses of aloes, or rhubarb with belladonna. A belladonna and ipecacuanha pill taken in the morning is often sufficient. The Hunjadi, Friedrichshall, or Pullna waters are sometimes useful. The diet, however, should be regulated so as to enable us to dispense with purgatives as much as possible.

The *treatment* of diarrhoea in cases of intestinal ulceration is a matter of great difficulty and often taxes our therapeutical resources to the utmost. The symptom must be treated at once, but not necessarily in the first instance by astringents.

If previous constipation, or if the appearance of the motions and furred tongue lead us to infer the presence of irritating material in the bowel, a dose of castor oil or Gregory's powder (Pulv. Rhei comp. P.B.) should be given first of all. In some cases a little grey powder (Hydrarg. c. Creta P.B.) and Dover's powder (Pulv. Ipecac. c. Opio B.P.) is to be preferred. Of astringents in the acute stages of the disease I think I might say that the remedy which of all others has best held its position is Bismuth. The sub-nitrate or carbonate of bismuth may be given, if the diarrhoea be not severe, in a mixture containing bi-carbonate of soda or prepared chalk. If the stools are more frequent and pain is present, some tinct. opii, or chlorodyne may be usefully added to the mixture. In severe cases twenty grains of the subnitrate of bismuth with five grains of Dover's powder every three or four hours forms a most valuable combination. This dose of bismuth is quite sufficient soon to infiltrate the excreta with the drug which may be observed to form a black pulverized residue in the utensil. One does not therefore see the use of giving larger doses which are apt to disorder the stomach. In more inveterate cases the compound kino instead of the Dover's powder may be given with bismuth. When the active symptoms subside, a little calumba or cascara or quinine may be usefully added to diminished doses of the previous drugs. We must at this point bear in mind the tendency in many cases for constipation to succeed diarrhoea.

This line of treatment will be found to be most generally useful in cases of diarrhoea of moderate degrees of intensity in which the colon is not extensively involved. In a much more limited number of cases sulphuric acid and opium seem to answer best. It often happens however that during convalescence a few drops of aromatic sulphuric acid in some simple bitter may be given with great advantage. A remedy which scarcely ever fails even in the worst cases to give relief, if only for a time, is the starch and opium enema of the Phar-

macopœia. Some acetate of lead may be added to this enema, or the lead and opium suppository may be employed with advantage. In more chronic cases and those of greater severity we still have a large armoury of more decided astringents to fall back upon, amongst which the lead and opium pill holds a high place. But the vegetable astringents kino, catechu, hæmatoxylum, tannic acid, may be each in turn tried in combination with opium with decided but often only temporary benefit. The aromatic chalk and opium powder of the Pharmacopœia given in a mixture containing tincture of catechu is a favourite remedy in these cases. Although all the vegetable astringents owe their efficacy to the tannin they contain, yet there is some peculiarity in each, and when one has failed another will often succeed, again in its turn to lose its virtues in the particular case. Almost all these cases require opium in addition to the astringent, and sometimes opium alone given in the solid form is the best remedy. This latter statement brings us to one that really should come first to the mind of the practitioner, viz., the necessity of insisting upon rest in the recumbent posture (best of all in bed) as of first importance in the treatment of this form of diarrhoea. The action of opium is mainly to keep the bowels in a state of physiological repose, and it is folly to disturb this action by jolting movements.

The diet should be regulated so as to give as little residue as possible for the bowel to digest. Meat essences, scraped meat, Liebig's Malt Extract, or Savory and Moore's infant and invalid food with or without milk, rice milk, albumen of egg diffused in milk and water, are suitable articles for the more acute stages and exacerbations of the disease. Stimulants should only be given sparingly, pale brandy is the best form, but in some cases port-wine seems to agree well. During convalescence, and on any threatening of a relapse the diet sheet must be carefully inspected. These patients suffer much from chilliness often alternating with heat, and precau-

tions should be taken to keep the extremities and abdomen well warmed. Hot poultices or fomentations over the abdomen are often serviceable and comforting. In some cases, however, the cold compress is preferable. Where much flatulence or pain are complained of, turpentine stupes are useful: small doses of animal charcoal given with bismuth give immediate relief in cases of flatulent colic. Dr. Pollock finds the application of a blister over the region of the cæcum coli to be sometimes useful in relieving pain and diminishing the severity of the diarrhoea. It is often a good plan when the patients begin to move about to apply a spongio piline compress to the abdomen to maintain the parts at rest.

I have dwelt at greater length than usual upon the special treatment of this distressing complication of pulmonary phthisis, not because I have had anything new to say about it, but rather because a necessarily large experience of such cases has prompted me to suggest those lines of treatment which I have found most successful at least in giving relief.

CHAPTER XIII.

TUBERCULAR MENINGITIS.

Tubercular meningitis,—most insidious in its supervention.—Illustrative cases exemplifying the most characteristic symptoms.—Comments.—Table of twenty cases showing prominent symptoms.—Conclusions from their treatment.

ALTHOUGH it might seem to the unreflecting scarcely fitting to treat of tubercular inflammation of the coverings of the brain in a work on chest diseases, a very little consideration will suffice to bring to mind the fact that this fatal malady most commonly occurs as a complication of phthisis, as part of a secondary general tuberculosis. I have already pointed out that tuberculosis is an affection which in no sense comes within the definition of phthisis but which very frequently arises as a complication of that disease.⁷⁷ When the meninges of the brain are importantly involved in this out-break of tubercle, the special symptoms that arise are so grave as to set aside from view all other conditions present.

There is no more striking feature about tubercular meningitis when it complicates phthisis than the uncertainty and insidiousness of its supervention. It is happily a somewhat rare disease, yet there is no case of phthisis, and no stage of any case in which the conditions for its possible occurrence are not present. A child may have had an apex catarrhal pneumonia resulting in some induration and flattening there but in complete subsidence of all symptoms, she daily improves, and leaving off all treatment, is regarded by her parents as well; but the doctor says that her chest is still delicate. She suddenly develops brain symptoms and is carried off in two or three weeks. This is a common history.

I will relate in a little more detail one or two cases which may be regarded as typical of their kind.—George

⁷⁷ Dr. Shepherd, *Gulstonian Lectures*, 1876, and *Brit. Med. Journal*, Sept. 1, 1877, also insists "on the pathological line which marks off general tuberculosis from ordinary phthisis."

B—— aged 12, a school-boy admitted into the Brompton Hospital under my care in August 26th, 1875. He was a thin, pale, somewhat underfed neglected-looking child, and his present ailment dated from an attack of measles four years ago, which was followed by a cough, the expectoration at that time being occasionally streaked with blood. The cough had remained more or less since. A month before admission to the Hospital he had kept his bed for a week with pain in the right chest, and since that time had been troubled with night sweats and loss of flesh.

At the time of admission the boy was but little troubled with cough; his tongue was slightly coated and bowels confined, but he had a fairly good appetite. The respiratory murmur was weakened over the whole chest and accompanied by slight occasional sibilus. The left base did not expand with inspiration and there was defective resonance on this side below the nipple level in front, and in the axilla, with more decided dulness below the same level posteriorly. On the right side there was dulness at the extreme posterior base for three fingers' breadth.

The patient was considered to be suffering from slight bronchitis with the remains of broncho-pneumonia affecting the left side principally.

There was nothing in the case to attract further notice until the 18th September when vomiting after food was complained of. On the next day the vomiting continued and there was some diarrhoea. On the 20th, pulse 84, respirations 20, temperature 99° at 8 p.m. Had vomited 6 times. 21st. No vomiting since last night, *urine phosphatic*, no albumen. The vomiting persisted through the 22nd, 23rd, and 24th, the tongue being white and coated with prominent papilla. *No headache or pain.* 25th, vomiting continues. Face flushed, skin hot, perspiring.

On morning of the 27th, patient was *unconscious* and could scarcely be roused to put out tongue, pulse 84. At 4 p.m. my

note was as follows. "Pulse 76, *irregular*, lips dry, tongue furred. Expression of face *drowsy, suspicious*. Will not answer questions. Tries to put out tongue when sharply told to do so, but fails. Is slightly delirious. Respirations 20 in the minute, temp., 100·8°. There is slight occasional *twitching of the left arm* and pectoral muscle. When aroused drinks oatmeal water with greediness. Evidently tries to answer questions put to him". 9 p.m.—Twitching of both arms and legs, picking at bedclothes, grasping at nothing. Has not vomited after taking cream and brandy, pulse 80 temp. 100°. Sept. 28, temp. 100·2° pulse 80, more restless. Fingers continually working scratching or pulling at teeth. *Muscles of back rigid.* 4 p.m. Movements of arms and legs more violent. Rigidity of back more marked; temp. 102·2°, bowels open from medicine, motions loose. 29th, temp. 101·2°, pulse 80, weaker. Slight external strabismus of left eye. 30th, 10 a.m., temp. 102·4°. 7 p.m. 103° pulse 120. Eyes roll slowly from side to side, pupils dilated. Oct. 1st, temp. 101·6° a.m. Oct. 2nd, temp. 101·6° a.m. Increasing emaciation. Some resistance to extension of arms. Conjunctivæ more sensitive, pulse 130 *regular*. Patient lingered for a week longer in much the same state and then sank.

Post-mortem.—The cerebral convolutions were found to be flattened, the pia mater over them congested. The ventricles were distended with clear fluid, the ventricular walls being soft, almost diffluent, the choroid plexus not congested.

At the base of the brain the pia mater was congested over the anterior and middle lobes, and presented innumerable miliary granulations. Over the central space between the middle lobes the membrane was thickened, opaque, and contracted. The left middle, and anterior lobes were intimately adherent at the fissure of Silvius by thickened, opaque yellow lymph, the veins in this fissure were blocked, and immediately under the pia mater the cerebral substance was softened down into a cavity. On the opposite side in the same position, the brain was much softened.

In this case during the first days of the attack no head-ache was complained of, nor did the boy at any time complain much of this symptom. Vomiting of food was the first, and for the first week, up to the period of commencing unconsciousness, was the only important symptom that might direct attention to the head. But in a boy it would first, at all events, direct attention to the stomach, and being associated with furred tongue and disordered bowels it was attributed to derangement of this organ. It, however, obstinately resisted all treatment. The first circumstance which lead me in this case to attribute the vomiting to its right cause was the observation by my clinical assistant that the urine which he had examined for albumen yielded on boiling a heavy cloud of phosphates. This was on the 4th day of the vomiting. Twitchings and irregular movements of the limbs with increasing unconsciousness supervened on the 9th day, with some rigidity of muscles, especially those of the back of the neck. The temperature which was not elevated at first was distinctly so from the 9th and 10th days. Squinting and latterly blindness were present in this as in many other cases of tubercular meningitis.

The following are the main features of a case which was under my observation in the Brompton Hospital from Dec. 12th 1876, and previously as an out-patient at Charing-Cross.

A man aged 33, predisposed to phthisis, had a cough with expectoration in February 1876, and in April began to suffer from fistula. He improved in health for a time, but the fistula continued to discharge freely, and in the late autumn the cough and expectoration were more severe, the hectic symptoms more marked, and physical examination shewed that he had sub-acute phthisis, affecting principally the apex of the left lung. At Christmas a sub-divided cavity was found to exist at this situation and the opposite lung, although enlarged, was partially consolidated at its summit. The pulmonary symptoms were now, however, quieter, and the patient referred his

slowly increasing debility to the discharge from the fistula. On the 10th of January weak iodine injections were used with the effect of somewhat lessening the discharge. Before the employment of these injections, however, the temperature had latterly been somewhat raised and the cough more troublesome. On this 10th of January the patient first complained during the night of slight headache and confusion of vision which was attributed to a small dose, $\frac{1}{100}$ th of a grain, of hyoscyamine given in a linctus at bed-time to relieve the cough, and which had caused some dryness of throat. But in the morning the pupils were not dilated. "Temp. high" pulse 100: cough lessened but expectoration tinged with blood. The hyoscyamine was repeated at night and gave much relief to the cough, but the headache returned with severity, and in the morning (12th) the patient vomited some bilious matter. Bowels freely opened, pupils not dilated, pulse 88. During the next two days the headache was slight, the tongue much furred, and patient vomited once with the cough.

On the 15th he was observed by my careful clinical assistant, Dr. Blackader to be "very nervous with much twitching of the limbs." Pulse 84. I saw the patient the next day at 5 p.m. and found the pulse 72 and regular; there was some twitching of the flexor tendons principally at the wrists. The manner of the patient was somewhat confused, and he complained of weakness and giddiness. There was now no doubt as to the diagnosis. On the 17th he became drowsy, the pupils were contracted, the left more so than the right. The headache was mainly frontal, the patient had some difficulty in selecting words to answer questions. A small dose of calomel had freely acted upon the bowels. Temp. 99.1°, pulse 60, respirations 18, slightly irregular. On the morning of the 18th, the respirations, 24 in the minute, were noted as being irregular, both in frequency and depth. Pulse 60, temp. 99.8°. Patient had been sick three times in the night and again this morning. The twitchings of the limbs, which had considerably lessened, came on somewhat severely for

half-an-hour this morning, but did not amount to convulsion. Evening temp. 99°, pulse 78, respirations 20. Jan. 19th, temp. 99.6°, pulse 84, respirations 26 more regular. Headache slight. On the 20th the morning temperature was 101.2°, pulse 94, respirations 26. Twitchings well marked. Bowels confined. In the evening, temp. 99.6°, pulse 78, respirations 36. Patient passed a sleepless night and was dull and drowsy the next morning. (21st). In the evening the temp. was 101.4°, pulse 92, respirations 30 somewhat irregular. He remained drowsy through the next day, increasingly so towards the evening. The temperature continued a little above 101°, pulse 93, regular, respirations 26. On the 23rd the morning temp., was 101.2° pulse 90, respirations 30. At 3 p.m. patient was "drowsy looking, but intelligent, complaining of headache; some hesitation in answering questions. Heart's action regular, tongue protuded evenly, no paralysis of face." No paralysis of limbs. Retina examined by Dr. Blackader with no positive result.

Jan. 24th, some irregularity of the pulse noted early in the morning, temp. 99.4° respirations 30. Next day (25th) at 10 a.m. stertorous breathing came on suddenly, patient aroused with difficulty, eyes opened when spoken to, no other sign of consciousness, temp. 101°, pulse 72, respirations 36. At 3 p.m. "respirations 28, irregular in depth: pulse 96, weak, regular, slight subsultus, patient difficult to rouse but complains of thirst." Evening temp. 101.4°, respirations 32, pulse 100, weak but fairly regular, increasing insensibility. During the 26th patient was restless appearing conscious at long intervals for a few moments only, urine passed under him, took nourishment with difficulty, temp. 100°, pulse 120, respirations 38. On the following day, both pulse and respiration became irregular, insensibility complete, at 10 p.m. marked lividity of face, pulse 138, respirations 40. Patient died at 2.15 a.m. on the 28th January.

The insidiousness of the onset of meningitis was well illustrated in this case. The symptoms were no doubt somewhat masked by the effect of the hyoscyamine. The furred tongue

rendered the headache and slight vomiting less noteworthy. The muscular twitchings which occurred on the fifth day rendered the diagnosis certain, and they continued more or less throughout the illness. The memory of the patient was markedly impaired, and he had a difficulty in finding words to answer questions which he evidently understood. The temperature was misleading as I have ever found it to be in tubercular meningitis, it was in this case up to the 12th day, lower than it had been the few days previously, and it never rose above 101.5. The pulse was moderately quick, and was on one or two occasions irregular. The respirations were at times remarkably irregular, both in depth and frequency although never assuming the Cheyne-Stokes type. Irregularity in respiration, and irregularity in pulse, the two having no definite relation to one another are common and important, but usually transient, symptoms in tubercular meningitis. I have never, however, observed anything approaching to the Cheyne-Stokes type of respiration in this disease.

Increasing drowsiness (with brief intervals of consciousness most difficult to explain), was in this as in all cases of tubercular meningitis the closing symptom, occurring intermittently at an early period, and gradually destroying consciousness entirely. Obstinate constipation, a common symptom in all head affections was not in this case noticeable until the eighth or ninth day, when the diagnosis was well confirmed.

The following table abstracted from notes of twenty cases that have occurred at the Brompton Hospital, which have been kindly collected for me by my friend Dr. Garlick a former clinical assistant there, shews pretty clearly the most prominent symptoms of tubercular meningitis when it arises as a complication in phthisis. Only those cases have been taken, of which the notes are sufficiently full, and in which, with one exception, the diagnosis has been confirmed by *post-mortem* examination.

N.B. *The figures placed to the left hand of the symptoms enu-*

merated refer to the day of appearance of the given symptoms. When figures are placed within brackets to the right hand of a symptom, they signify its duration in days.

CASE.	SYMPTOMS IN ORDER OF OCCURRENCE.	REMARKS.
(1) M. æt. 12.	1. Vomiting with diarrhoea. 8. Phosphatic urine. Deepening coma. 14. { Irregular pulse 84. Temp. 100°—102°. Delirium. 15. { Muscular twitching. " rigidity. 22. Death.	
(2) F. æt. 20.	1. Headache (5). 2. Ptosis R. eye with dilated pupil. 6. Delirium (3). 9. { Deepening coma. { Retention of urine. 14. Stertorous breathing. 15. Death.	Chiefly frontal. Temp. not taken, pulse ranged from 80—90 until last two days when 100—110.
(3) F. æt. 15.	1. { Intense headache. Vomiting with furred tongue. Pulse irregular, intermittent. Deepening coma. 15. { Slight convulsions of upper extremities. Ptosis, left. 20. { Stertorous breathing. { Death.	
(4) M. æt. 33.	1. Heaviness, confusion of ideas. 2. { Retention of urine. { Coma. Dilated pupils. 5. Death.	
(5) M. æt. 28.	1. Feverishness. 3. { Heaviness and stupidity. { Coma. Temp. 99°—100°. 4. Death.	Death 36 hours after pronounced symptoms.
(6) M. æt. 18.	1. Pain right side of head with numbness and tingling in left arm and cheek (10). 10. Paralysis left side of face, partial hemiplegia with diminished sensations. 14. Stupor. 17. Coma, pulse 80 steady. 29. Death.	P.M. Tuberclie in pia mater. Extensive cerebral softening.

CASE.	SYMPTOMS IN ORDER OF OCCURRENCE.	REMARKS.
(7) M. æt. 41.	1. Headache with foul tongue (13). 2. Difficulty in answering questions. 3. Delirium, obscured intellect, P. 68. 13. Increasing stupor, P. 48. 14. Coma, contracted pupil, P. 84. 16. Death.	
(8) M. æt. 33.	1. Sudden giddiness and incoherence. 4. { Epileptiform convulsions. 9. Coma. 9. Death.	Passed off but left patient weaker.
(9) F. æt. 27.	1. Pain back of head. 1. { Confused answers to questions. 1. Constant sickness. 1. Confined bowels. 13. { Commencing coma. 13. { Pupils fixed, pulse slow, full. 14. Convulsions. 16. Death.	No headache complained of in this case. Admitted with these symptoms.
(10) M. æt. 25.	1. Headache, (7). 7. Sudden coma with stertorous breathing, no paralysis. 8. Death.	
(11) F. æt.	1. Impaired memory. 3. Convulsions and delirium, stertorous breathing. 4. Death.	Convulsive attacks were very prolonged and attended with screaming.
(12) M. æt. 28.	1. { Intense headache with mania. Tongue clean. 1. Partial right hemiplegia with paralysis and anaesthesia of right half of face, P. 64, T. 98.2°. 2. Semi-unconscious. Restlessness, picking bed clothes. 4. { Coma absolute with small contracted pupils, (1 hour), P. 120, R. 76, T. 101.2°. 4. Death.	
(13) M. æt. 25.	1. Vomiting (14). 4. Persistent headache (10). 14. Pupils normal, Temp. 101°—102°. 16. Right hemiplegia. 9 am. Pupils normal, sensitive, P. 66. 10.30 am. Coma, P. 80, R. 40, Temp. 101.2°. Optic discs normal.	

CASE.	SYMPTOMS IN ORDER OF OCCURRENCE.	REMARKS.
	17. Death.	P.M. A yellowish tumour size of a pea in brain substance near anterior part of R. corpus striatum.
(14) M. æt. 38.	1a. Headache (1 mo.) 1. Bowels costive, p. 120. 3. Dilated pupils (4). 4. Paralysis of 3rd on right side. Dilated insensitive pupils. Incoherence. 6. Paralysis of 3rd on left side. Delirium, Coma. 7. Death.	Headache persistent more or less.
(15) M. æt. 32.	1. Sickness (2). 2. Drowsiness, bowels confined. { Left internal strabismus. 7. { Coma, P. 60. 8. Delirium, incontinence of urine. Left pupil dilated, insensitive, P. 52. 9. P. 48, R. 44, violent delirium during the night. 10. Convulsions. Complete coma with stertor. 11. Death.	
(16) M. æt. 32.	1. { Intense headache (3). { Drowsiness, slow pulse. 4. Muttering delirium. 5. Coma no paralysis, P. 70—104, R. 26—24, T. 99·2°—98·4°. Pulse irregular in time. { Pulse irregular. 6. { Resp. ditto. { Paralysis of 7th. 7. P. 120, R. 4, T. 98°. Right pupil dilated. 8. Death.	Had complained of headache recurring at intervals for a month previously.
(17) M. æt. 31.	1. { Headache and sickness (4). { Skin cool. 3. Temp. 98·8°, bowels open. 4. Occasional delirium. 5. Delirium alternating with coma, no change in optic discs, pulse 68, Temp. 100·8°. 6. Consciousness improved, gives rational answers, P. 84. 9. No paralysis, answers questions. 10 p.m. Death.	3 or 4 inspirations followed by a pause. No P.M.

CASE.	SYMPTOMS IN ORDER OF OCCURRENCE.	REMARKS.
(18) M. æt. 44.	1. { Headache with confined bowels. 2. { Coma with stertor (2). Pupils normal and sensitive. 3. { Muscular twitchings and rigidity. 3. { Temp. $101\cdot3^{\circ}$, P. 104, R. 38. 3. { Pupils widely dilated. 3. { Death.	Duration of head symptoms barely 48 hours.
(19) M. æt. 33.	1. Headache slight with compound vision. 3. Bilious vomiting, bowels open, pupils normal, pulse 100. 5. Muscular twitchings, pulse 84 regular (4). 7. Drowsiness, hesitation in answering questions, Temp. $99\cdot1^{\circ}$, P. 60, R. 18, irregular. 14. Pulse irregular for a few hours. 15. Coma. 17. Pulse and Resp. irregular. 18. Death.	P.M. purulent lymph all over surface and base of brain. Turbid serum in ventricles.

The following conclusions which I will put in the form of propositions will be found in accord with the facts enumerated in the above table, and will I think embrace what is reliable in the diagnosis of this complication, often so startling in the suddenness of its onset.

(1). *Persistent headache and vomiting are the most common first symptoms of the disease. They may or may not be combined. They are usually associated with furred tongue and disordered bowels, which tend to mask their significance.*

The headache of tubercular meningitis does not affect with constancy any particular portion of the head. It is sometimes frontal, often over the crown of the head, occasionally at the back or on one side. Although always a sign to cause anxiety when it occurs at all severely or persistently in phthisis, yet it is never, even when associated with vomiting, sufficient to enable us to form a diagnosis. In several cases of phthisis, I have found headache so severe, persistent and, taken to-

gether with the general aspect of the patient, so apparently characteristic of meningitis, as to have led me to feel confident as to its real significance—yet again and again my suspicions have proved to be unfounded. On the other hand, in the majority of cases of true tubercular meningitis that have come under my observation, there have been, as in the two cases above related, attendant phenomena to otherwise explain the headache until the appearance of more decided signs removed all doubt. Hence *headache or vomiting, although not sufficient for diagnosis are signs which if not readily relieved by treatment should always arouse grave suspicions.*

(2). *Disordered vision, impaired memory and confusion of ideas are signs which taken in association with headache are almost diagnostic. Muscular twitchings (and of course convulsions) absolutely so.*

Any or all of these signs may, as will be seen by a glance at the table, closely follow the appearance of headache or vomiting. They may, one or more of them, constitute the first symptoms of the disease. Paralysis of the third or sixth nerve usually occurs among the later symptoms, when the effused lymph and contractile tissue drags and exercises pressure upon the nerves. Ophthalmoscopic examination of the retina may yield a valuable positive result, but the absence of granulations upon the retina signifies little.

(3). *Drowsiness deepening into coma, but often with intervals of consciousness, is the most constant symptom of tubercular meningitis. It depends upon effusion into the ventricles, and thus is among the final symptoms.*

(4). *Irregular pulse, irregular respiration and excess of phosphates in the urine, are amongst the occasional signs of tubercular meningitis.*

Both irregularity of the pulse and of the respiration are not uncommon after the period of coma, but they are then signs of little importance. A marked irregularity of the pulse occurring, however, early in the attack is I think of greater significance than is usually recognised. The pulse is commonly rather slow than quick, sometimes markedly infrequent.

The respirations are rarely affected during the early periods of the attack.

(5). *The temperature is as a rule not much elevated in these cases of tubercular meningitis. It is more often raised towards the end of the attack, its rise being apparently associated with secondary inflammatory lesions. It is of little or no value in the diagnosis of tubercular meningitis.*

In the first place in the cases under consideration we have already a cause of possible elevation of temperature in the pulmonary disease present, and secondly, the temperature is not usually high in tubercular meningitis. On this point, however, the cases in the table do not yield sufficient data but the two cases above related and also cases 1, 12, 13, 17, tend to shew, contrary to the teaching of Wunderlich, that the temperature rather tends to rise than to sink as the fatal termination draws near.

Excess of phosphates in the urine may be a valuable sign in helping us to interpret the earlier symptoms of tubercular meningitis. In one case already mentioned, it certainly led me to a right diagnosis some days before other absolutely certain signs presented themselves. There was no such excess present, however, in another case⁷⁸ so that this sign like some others will probably turn out to be of importance, when present, but not essential.

The duration of this disease is various, it may terminate in a few hours, or days, or weeks.

All treatment, after the diagnosis becomes certain, is hopeless, save for the purpose of giving relief to suffering. A brisk calomel purge and the steady application of ice or of an iced water cap to the shaven head are the best means of affording relief. Leeches to the temples are sometimes

⁷⁸ I am here referring to the rude but facile test of obtaining a cloud on boiling which clears up on the addition of acid. Perhaps a more elaborate chemical analysis might be useful in such cases but it could only be carried out in the laboratory.

serviceable, and blistering fluid applied in strips over the crown of the head appears in some cases to give ease. The room should be darkened. Care must be taken that the bladder be relieved if necessary, and the wants of the patient in the shape of food and drink supplied by a watchful nurse.

PART II.

CHAPTER I.

BRONCHITIS—BRONCHIAL CATARRH.

DEFINITION—Etiology, age, sex, occupation, changes of temperature.

“Catching cold,” mechanism of: dusty employment: exanthemata: Influenza, hay asthma: dentition. Hereditary influence, heart diseases. Morbid appearances, a sample often seen during life in the trachea—Three stages of catarrh, congestive, transudative, secretory—Tendency of acute catarrh, (1) to recovery, (2) to become chronic, (3) to purulent inflammation, (4) to extension to deeper parts: 1 and 2 most common—Table summarising the history, distribution and tendencies of chief varieties of bronchitis—Symptoms of acute catarrh of larger tubes: physical signs—Catarrh of smaller tubes, symptoms, physical signs—Diagnosis of bronchitis, from pulmonary oedema, tuberculosis and acute phthisis—Prognosis—Treatment of capillary bronchitis in the adult; in infants and old people—Treatment of chronic bronchitis—Secondary bronchitis not separately considered—Remarks on dust bronchitis and on the effect of inhaled irritants in causing phthisis: dust, cold and damp compared in their effects upon the bronchial tubes and lungs. Inhaled dust may produce catarrhal phthisis, chronic bronchitis with or without asthmatic paroxysms, or indurative phthisis—Illustrative case of dust bronchitis—Remarks.

Acute Bronchitis is an acute catarrhal inflammation of the mucous membrane of some portion of the bronchial tract.

This, in our English climate, very common disease may attack any individual at any age, liability to the occurrence of bronchitis being influenced by age, sex, occupation and condition of life, chiefly in so far as these circumstances favour exposure to the known exciting causes of the disease, or render the individual less able to resist the action of such causes. Thus young children and old people are most prone to bronchitis, and for the same reason the disease is apt to run at such periods of life a graver course. The male sex is more exposed to the causes of bronchitis than the female, and suffers more accordingly.

Of all the causes of bronchitis, depression of temperature is the most important. The attack commonly supervenes upon exposure to sudden changes of temperature, or to wet cold winds, especially in depressed conditions of the system, as after being over-heated by exertion, or exhausted by mental fatigue or shock. Dr. Sturges¹ finds from his careful analysis of the Registrar General's returns for a series of years, that cold is a far more potent cause of bronchitis than of pneumonia, January being the month of highest mortality from bronchitis. A large rainfall again, Dr. Sturges finds, increases the mortality from bronchitis, but does not affect the pneumonia rate. Bronchial catarrh is very prevalent in low-lying marshy districts, cold wet weather with variable winds being most favourable to its occurrence. "Catching cold" has been explained by Riegel in accordance with the experiments of Rosenthal, who found that the superficial vessels of animals exposed to a high temperature became dilated and partially paralysed. Such animals on being removed to a cold room lose heat rapidly, their temperature becoming quickly reduced to below the normal by the rapid radiation of heat from the blood coursing through the surface vessels. Whether the cold be "caught" in the head, or in the bronchi or lungs, or in some other internal organ is, in part at least, a matter of constitutional predisposition; but it must be borne in mind that the small vessels and capillaries of the air passages are, in direct proportion to their depth from the surface, exposed to the same effects of heat and radiation as those of the cutaneous surface.

It cannot, however, be doubted, that catarrhal inflammation may arise in quite an opposite manner, viz., by the constriction of one set of superficial vessels, causing engorgement of another and a deeper set. Thus the playing of a cold draught upon one section of the branches of the carotid vessels distributed to the neck, will cause active congestion of corresponding

¹ On Pneumonia, p. 153 *et seq.*

internal parts, giving rise to pharyngeal or laryngeal catarrh, or inflammation. This is but the reverse action to that of a blister or poultice, which, by causing engorgement or inflammation of the external surface, relieves congestion of the deeper parts.²

The effect of prolonged chill to the feet in giving rise to the same phenomena of throat or chest catarrh, is equally well known but much more difficult to explain. Ice to the head will lower a febrile temperature and doubtless, in susceptible persons, it might depress the normal temperature, and the same result may follow the exposure of the lower extremities to continued cold. It would seem to follow, however, from the recent observations of Mr. Knowsley Thornton³, upon the effect of the application of an iced water cap to the head in reducing temperature, that the antiphlogistic action is brought about in a great measure through nervous influence.

Dusty employments lead to chronic catarrh of the proximal air passages, and if long continued, such catarrh may extend more deeply into the lungs. It is very instructive to note, however, how out of those who are exposed to obviously mechanical influences of this kind, many escape unharmed. In noting the history of patients who have been suffering from irritative bronchitis, I have found instances in which the fathers of the patients had passed their lives at the same occupation without complaint. The second or third generation seems to become more vulnerable to a given influence.

Many a fresh catarrh in cases of confirmed pulmonary disease, may be traced to dusty winds, or to the irritating fogs which occasionally prevail in this metropolis. Blood inhaled to distant bronchial tubes during an attack of hæmop-

² I am aware that this illustration will not be accepted by some physicians who deny the efficacy of both the remedies named, remedies which are, nevertheless, to my mind abundantly established amongst those best known to experience, and most reliable in practice.

³ *Medico-Chirurgical Transactions.* 1877.

tysis, or from haemorrhage during tracheotomy, may set up bronchitis.

In certain exanthematous diseases, especially in measles and in a minor degree in small-pox and syphilis, the mucous membrane of the air tubes seems to partake in the specific eruption. Acute bronchial catarrh commonly supervenes upon or forms a part of the epidemic disease Influenza. Perhaps in this category Hay Asthma should be included as a specific catarrh due more distinctly than influenza to the reception of a particulate poison. Finally, many children during the first dentition suffer repeated attacks of bronchial catarrh, coincident with the eruption of each tooth; just as other children suffer at these times from nasal catarrh, others again from intestinal catarrh. During this period of dentition, children are frequently the subjects of irregular febrile disturbance, and during the febrile periods they are more liable to chills; but for the most part the catarrhal affections prevalent at this age cannot be explained otherwise than as being of reflex nervous origin, and the fact that under the same circumstances we may get convulsions in the place of either of the above occurrences is strongly confirmatory of the truth of this hypothesis.

Recurrent bronchial catarrh, associated with asthmatic symptoms, is well known to be an hereditary affection, and may occur very early in life. The winter bronchitis to which many individuals become subject at certain periods of life is also very markedly hereditary, and is no doubt really a phenomenon of senility. It cannot with these exceptions be said that bronchitis is hereditary. Certain heart diseases, mitral regurgitation, and in a still greater degree mitral stenosis (a disease, I am persuaded, often congenital,) predispose to bronchial-catarrh by obstructing the return of blood from the lungs and by so causing mechanical congestion of these organs and of the small bronchi. Even the larger bronchi are not exempt from this influence, for, although they have their own system of veins, yet if the circulation through those

branches of the bronchial artery which open into the pulmonary veins is hindered, it is obvious that there must be a stress of circulation through those which open directly into the bronchial veins, and they accordingly are apt to become engorged. Finally it is very important to remember that one attack of bronchitis predisposes to future attacks.

The morbid appearances of simple catarrh are but rarely to be observed *post-mortem*, but they may sometimes be seen in perfection in the trachea by means of the laryngoscope during life. These appearances consist of hyperæmia, bright redness, with minute vascular injection, and swelling with undue lacerability of the mucous membrane, upon the surface of which, after the very first stage, there is an excess of clear or opaque mucous secretion. An increase of cell elements of the sub-mucous and epithelial tissues is to be observed with the microscope. The hyperæmia entirely subsides after death, but in some cases of very severe character, minute haemorrhages into the mucous membrane have been observed. On the affection becoming chronic, the redness becomes more dusky, more slaty in hue, the mucous membrane thickened and toughened. The phenomena of catarrh of the bronchial are identical with those observed in other mucous membranes and are referable to three stages, viz. (1), that of redness and swelling of the membrane with arrest of secretion, (2), that in which there is a transudation of serous fluid mingled with epithelial sheddings and mucous elements, (3), the mucous or pus elements increase and cause the secretion to become thicker and more opaque. For some time after the inflammation has subsided the mucous secretion from the surface is excessive from hyper-vascularity of the glands.

The tendency of acute catarrh is, 1, to complete recovery, 2, to become chronic, 3, to purulent inflammation, 4, to spread to deeper parts. The first and second are of most common occurrence, the last is rare.

In the subjoined table will be found summarised the history, distribution, and tendencies of the chief varieties of bronchitis which will serve to prompt, and in part to supplement further remarks. To discuss each variety and subvariety separately, and in full, would take up a volume and involve much repetition.

The *symptoms* of acute catarrh affecting the large bronchial tubes are tolerably familiar. They usually commence with those of an ordinary cold in the head, chills of a creeping character, never amounting to rigor, occurring from time to time on the first day, and being attended with a feeling of malaise, a somewhat hurried pulse, slight soreness of the throat, sneezing, and coryza. The temperature is raised a degree or so above the normal, but although there is thirst and a considerable sense of feverishness, the febrile phenomena are really very slight in adults; more decided in young children. After some twenty-five or forty-eight hours the patient complains of a soreness or rawness, as it is usually more accurately described, felt behind the upper sternum. The cough is frequent, dry, and attended with more or less pain of a rending character behind the sternum. A sense of constriction or oppression is complained of in this region, and the breathing is perceptibly quickened. The voice is deepened and sometimes husky or suppressed. On the second or third day secretion takes place and with the expectoration of a thin aërated mucus the patient soon experiences a marked sense of relief. In fact the pyrexial stage has already passed, the pulse is quiet, the cough loose, and expectoration easy, the mucus expelled becoming more opaque and semi-purulent. The secretion subsides, and the cough gradually lessens and usually disappears in a week or ten days, but in the morning, after his first sleep, the patient still feels some oppression in the chest and is not relieved until he has brought up some purulent mucus.

As regards physical signs we may hear a few dry, sonorous

SUPPLEMENTARY TABLE OF THE ASSOCIATIONS AND PHENOMENA OF BRONCHITIS.

BRONCHITIS.	ASSOCIATION.	EXCITING CAUSE.	AGE AT WHICH MOST PREVAILS.	DISTRIBUTION OF SIGNS.	COURSE.	TENDENCY.
1. PRIMARY	Simple	Exposure to cold	Youth and age	Double basic	Acute	To recovery or death
2. SECONDARY	(1.) Gout and kidney disease (2.) Alcoholism (3.) Heart disease (4.) Phthisis (5.) Mechanical irritation (6.) Asthma (7.) Croup	(1.) Defective elimination (2.) Elimination of drug and extension from throat catarrh (3.) Mechanical congestion of bronchial membrane (4.) Chill, presence of "tuberclercle" (5.) Dust inhalation (6.) Afflux of blood to lungs and bronchi during ineffectual inspiratory movements (7.) <i>a.</i> Ditto. <i>b.</i> Specific inflammation of membrane	(1.) 30 to 50 (2.) 20 onwards (3.) Before 35 (4.) Variable (5.) Variable (6.) Before middle life (7.) Young children	(1.) General bi-lateral (2.) Larger tubes (3.) General (4.) Local one-sided (5.) General (6.) Bases (7.) Bases		
3. RECURRENT	(1.) Commonly hereditary, emphysema, spasm of tubes (2.) Hay asthma	(1.) Exposure to cold .	(1.) Middle and advanced life (2.) Youth onwards	(1.) General, principally bases (2.) Larger tubes	Acute	(1.) Chronic (2.) { Recovery on removal from locality.
4. CACHECTIC	(1.) Simple malnutrition, over lactation (2.) Tubercular or (3.) Scrofulous diathesis	{ Exposure	Before middle life	(1.) Apices (2.) One apex and (3.) General	Acute { Acute or Chronic	Phthisis. { Chronic

râles vibrating through the chest, but these are obviously produced in the larger tubes, and if only the largest bronchi be affected no râles of any kind are heard.

When the acute catarrh affects the *smaller and capillary bronchial tubes* the dyspnœa becomes marked, with lividity of lips and an anxious distressed expression of face, the nares expanding with each respiration. In feeble and old people, there may be considerable systemic shock with general prostration, reduced temperature, and rapid feeble pulse. In more robust persons the febrile action is at first decided, although the temperature rarely mounts above 101° or 102° the pulse is full and not very quick, between 80 and 100 in the minute, the respirations being rapid, out of proportion to the pulse. The cough is frequent, and is very soon accompanied with the expectoration of a viscid adhesive mucus, difficult to get rid of. The digestive functions are impaired, the tongue furred, bowels usually confined, and the urine loaded with lithates.

On inspecting the chest, the thoracic movements are observed to be increased both in frequency and depth, the diaphragm partaking but little in the respiratory efforts. The explanation of this is soon found, for it is with the front and upper parts of the lungs that the patient is principally breathing. The resonance on percussion is everywhere unimpaired, it may be even increased. Fine bubbling râles are audible over both posterior bases: to a much less extent, or not at all over the upper and anterior portion of the chest where, however, sibilant and sonorous râles prevail. This distribution of the râles in bronchitis is mainly a question of gravitation, and the reason why the respiration in such cases becomes thoracic rather than diaphragmatic is plain. Dr. Walshe has well observed that even in bronchitis of mild type and not involving the capillary tubes, we may still get fine bubbling râles at the posterior bases from gravitation of the secretion to the minute tubes.

The *diagnosis* of bronchitis of this degree depends upon the

symmetrical distribution of fine bubbling râles not associated with any percussion dulness, or bronchial quality of breath-sound, and with but a moderate rise of temperature.

The diseases which may be confounded with it, are pulmonary œdema, tuberculosis and acute phthisis. If œdema be limited to the lungs, however, it must be dependent upon cardiac defect, the history and signs of which must be looked for. Tuberculosis is the disease most readily to be mistaken for bronchitis, for the *signs* of acute pulmonary tuberculosis are almost identical with those of capillary bronchitis. If the tuberculosis of the lungs be only a part of a general distribution of tubercle, the special adynamic symptoms of that disease will be apparent. When restricted, or mainly so, to the lungs the excessive dyspnœa and great prostration occurring in a patient in early adult or middle life are suspicious signs. The temperature may not help us at all, for in some cases of pulmonary tuberculosis it is not high. But, if present, a high temperature, especially when accompanied by profuse sweatings, always means something more than simple bronchitis. The distribution of physical signs is, however, in tuberculosis somewhat different, and their characters are not quite the same. With urgent dyspnœa we may have comparatively little secretion sounds and these are quite as marked at the apex as at the base, and sometimes more so. There may be at one apex some evidence of previous pulmonary disease. With only scattered sonorous or sibilant râles on the chest, or these accompanied with a short inspiratory crepitus, we have, in tuberculosis, quite a disproportionate amount of dyspnœa, with marked recession of soft parts. The history of attack and other features of the case, however, exclude asthma.

Acute phthisis cannot, after attentive auscultation and percussion, well be confounded with bronchitis. More or less pneumonic crepitation with patchy tubular breathing and larger clicks are superadded to the catarhal bronchial râles

and the signs are distinctly more advanced at some one portion of the chest, whether this be the apex or base. The fever again is characteristically high and remittent.

The *prognosis* in bronchitis of whatever degree, is, in adults, most generally speaking, favourable. Capillary bronchitis in very young and in old people is, on the other hand, very frequently fatal, yet there is perhaps no disease whose mortality is more influenced by treatment than that we are now speaking of.

Taking capillary bronchitis in the adult as our type in regard to *treatment*, the first thing to be seen to is the warmth of the room: a fire and a steam kettle are the first things to procure, so as to raise the temperature of the room to between 65° and 67° F., and immense relief is given to the patient by this means alone. Due care must be taken, however, to ensure a proper supply of air as well as to preserve a uniform temperature. The use of the steam kettle is not only to moisten the air of the room but in most bed-rooms it is the only possible means of raising and maintaining the temperature at the desired height. A large mustard or mustard and linseed poultice should be applied to the front of the chest, or to the back, and followed up by hot linseed applications or cotton wool to the chest. In children a jacket poultice is often very useful, but one must not forget that both in young or weakly children, and in old people, a linseed poultice wrapped round the chest may be a very serious impediment to free thoracic movements, and in such cases it is often more judicious to have recourse to cotton-wool covered with oil silk and an occasional mustard, or mustard and linseed poultice to keep the blood determined to the surface.

As regards drugs, a saline mixture with ipecacuanha is the best to begin with. In strong adults antimony wine is very useful. In old people, on the other hand, we must usually add ammonia to our prescription. The special danger in infants arises from the possible occurrence

of pulmonary collapse and secondary broncho-pneumonia. The collapse arises from want of power of expectorating, and the resulting broncho-pneumonia is just as mechanical in its origin, being dependent upon the aspiration of blood, during the thoracic efforts at expansion, to those parts of the lung which cannot expand on account of their bronchi being plugged. The timely administration of ipecacuanha emetics, if the secretion be abundant will avert this danger. Friction with stimulating liniments such as the ammonia or acetic turpentine liniments of the Pharmacopœia, if necessary further diluted, is of great service in young children after the first stage has passed.

In old people danger arises principally from exhaustion or paralysis of the bronchial tubes the latter, perhaps, being but another evidence of exhaustion. To avert these dangers, we must, from the first, support the patient by the frequent administration of nutritious liquids, and by the timely employment of stimulants in carefully regulated doses. Of all alcoholic stimulants, brandy is certainly the best for this purpose. The administration of opiate remedies in bronchitis should, as a rule be avoided, and absolutely so in cases in which lividity of lips shows already defective aeration of blood. In young children and old people opium must be used for bronchitis with the utmost caution. When the heart fails, and symptoms of over-loading of the right ventricle present themselves, digitalis may be usefully given, stimulants persevered with, and dry cupping may be tried with advantage. In a few cases, I have certainly seen good results from the substitution of belladonna for digitalis. As a sedative at night, bromide of ammonium with aromatic spirits of ammonia is one of the best we can choose. Chloral is not very suitable in acute cases, but a small dose combined with bromide of ammonium will suffice to give rest without risk of depressing the heart's action too much.

Next to avoiding a fatal issue, our efforts should be directed

to prevent the case going on to chronic bronchitis, especially in those who have had previous attacks. When the acute stage is past, some patients at once convalesce without any special treatment. In other cases the secretion continues abundant and more purulent. The saline mixture must now be given less frequently, or changed for a more stimulating expectorant containing senega and ammonia, and some mineral acid with calumba or quinine, ordered to be taken twice a day. It is often judicious to leave the bronchial mucous membrane alone, and to direct treatment towards improving digestion and appetite. Hence, in such cases, the gum resin expectorants are better avoided. Poultices have, of course, by this time been omitted, and cotton-wool applications substituted for them. The turpentine acetic liniment is of great value in this stage, its usefulness being partly, I suspect, due to inhalation of turpentine vapour.

In all cases of secondary bronchitis we must direct our treatment with due regard to the removal or amelioration of the more general disease which underlies the bronchial affection. Cases of secondary bronchitis are, as a rule, chronic, with acute exacerbations.

With bronchitis secondary to gout or kidney disease, to alcoholism, to heart disease, and to already existing diseases of the lungs I will not further deal. The affection of the bronchial mucous membrane is, in such cases, most commonly a part of a much wider affection. It is the primary disease that we should especially treat, and to direct too much attention to the pulmonary condition would apparently sanction an erroneous view of such cases.

I will now venture to make a few general remarks, such only as seem warranted by my comparatively limited experience, respecting those forms of chest disease which arise from the inhalation of dust, and will then proceed more particularly to describe a case of dust bronchitis.

Although dust-phthisis has attracted much notice, dust-bron-

chitis is perhaps the more important affection, since it is of far more frequent occurrence and most commonly forms an early and remedial stage of the more grave disease.

The effect of inhaled irritants upon the lungs and bronchi, must be considered in more than one aspect. It does not follow that because a man is a potter or charcoal burner and has phthisis that, therefore, he is suffering from potter's or charcoal burner's phthisis. True, or rather typical irritative phthisis is rarely seen in London, but at the Brompton Hospital we frequently meet with patients who have followed dusty occupations, who present the characteristic signs of advanced phthisis with excavations and softening on both sides, and with, it may be, laryngeal complication, yet who present none of the peculiarities—evidences of induration and contraction of lung and pleural thickening—which mark cases owing their origin to inhaled irritants, although their histories are sufficiently significant to show that the occupations they have followed have had something to do with the incitement of their lung disease. Making all allowances for the fact which I have already much insisted upon, *viz.*, that as the disease advances, all varieties of chronic phthisis tend to merge into one type in which havoc of lung and suppurative fever are the dominant features, one cannot assert that occupation has, in these cases, done more than produce the preliminary catarrh which, in persons predisposed to phthisis, has developed the first inflammatory changes of that disease. Cases of phthisis traceable to dust are really very common, and do not necessarily present any clinical features which would lead us to suspect their true origin. Many such cases, if their true origin be early recognised, may be saved by timely removal from their employment.

In truth, as I have already much insisted upon and as Niemeyer more strongly held—bronchial catarrh is the beginning of the majority of cases of phthisis. Whether the irritant be dust or cold wet air, a greater or less degree or duration of

exposure to it suffices, in those predisposed, to develope the pulmonary disease. We would not call these cases dust-phthisis or damp-phthisis or cold-phthisis, still less potter's dust phthisis or miller's or stone-mason's dust-phthisis nor dry or wet cold-phthisis, but we rationally include them all under the term *catarrhal phthisis*, and as such they require no further comment here.

It would seem that in those cases in which phthisis at last develops with signs characteristic of its irritative origin, the patients have tolerated the dust-laden atmosphere in which they have lived for a longer period, until the lung structure has become largely permeated with foreign matters. The first thing that strikes the observer of cases of true dust-phthisis is that the conformation of chest is not that of the ordinary phthisical invalid. Large broad chests with limited flattening and impaired movement at one apex are the features most usually noticeable.

In other cases again, the whole effect of the irritant falls upon the bronchi. Habitual cough with recurring bronchitis and subsequent emphysema, paroxysms of asthma, &c., are the leading phenomena in these cases. They are the most common of all occurring amongst potters, miners, flax-dressers, millers, plasterers, masons, and others.

It seems thus to be true to say that the inhalation of dust particles may produce three sorts or degrees of chest disease.

(a.) It may produce catarrhal phthisis not to be distinguished from other forms of catarrhal phthisis.

(b.) It may produce chronic bronchitis and emphysema, with or without asthmatic paroxysms, and which may or may not (as with chronic bronchitis originating from other causes) lead ultimately to phthisis.

(c.) It may, with comparatively little preliminary suffering of a bronchial kind, set up indurative pulmonary disease.

Are not these, however, types of pulmonary affection which we have already described under the terms catarrhal

and fibroid phthisis, and have yet to refer to under the heading, chronic bronchitis. There is, clinically speaking, no essential difference between them, but in their beginnings a more particulate irritant is concerned which a little cotton-wool might arrest by filtration, and in the ultimate dissection of the most striking variety amongst them such dust particles may, if inorganic, be recovered from the lungs by incineration.

The following case is not an unusual one of bronchitis and asthma induced by the inhalation of dust.

William C—, aged 38, married; residing in London, admitted into the Brompton Hospital under my care January, 23rd, 1877. Patient was a man of temperate habits, married, one child living: his wife had had one miscarriage, but there was no history of syphilis. He had been for twenty years employed as a fret-cutter. Four years previous to admission, he had had an attack of bronchitis, and since that time he had suffered constantly from cough, with suffocative attacks nearly every night. Patient had never had haemoptysis but had lost flesh considerably. His father died of rheumatic gout, and his mother of dropsy. There was no phthisis in the family. On admission, patient complained of cough, worse at night, and of severe attacks of dyspnoea at night. Expectoration moderate. Occasional night-sweats. Appetite poor, bowels constipated.

He was a tall and fairly well-built man with a somewhat suffused countenance and breathless look. The chest was well formed, but expansion with inspiration impaired. Right semi-circumference, an inch and a half above the nipple level, $15\frac{1}{4}$ inches, left $15\frac{3}{4}$ inches. Right ditto, two inches below nipple level, 16 inches, left $16\frac{1}{4}$ inches. Percussion note over the front of the chest was hyperresonant, the resonance extending on the right side a hand's breadth below the nipple, on the left side over the normal area of heart's dulness, and inferiorly to the costal margin. Diffused sibilant râles were audible front and back, and at the posterior bases

some mucous râles were heard. At the time of my visit on the 26th of January, the patient had been in Hospital three days and was suffering, as before admission, from nightly attacks of dyspnœa, and from troublesome cough. He was on the ordinary full diet of the Hospital. The *Mistura Potassii Iodidi cum Stramonio* of the Hospital Pharmacopœia, containing 3 grains of iodide of potassium, and $\frac{1}{4}$ gr. of extract of strammonium to each dose was now ordered to be taken at 12 noon, 4 p.m., 8 p.m., and 12 midnight, the diet remaining the same, and from the next night he had no serious dyspnœa. On January 31st, a note is entered. "Patient feels much better, and dyspnœa almost gone. Cough easier." February 3rd, "has not felt suffocating sensation for the last six nights." He continued the mixture, however, for a month, and then took it in half doses for another fort-night. On February 8th respirations were free and unaccompanied by râle, and by the end of the month the cough had disappeared.

March 1st. Right semi-circumference above nipple, $15\frac{1}{8}$ in. left, $15\frac{1}{2}$. Right semi-circumferences below nipple $16\frac{1}{3}$, left $16\frac{1}{3}$.

Expansion above nipple level, ordinary inspiration $\frac{1}{8}$ th inch: deep inspiration $1\frac{1}{8}$ to $1\frac{1}{2}$ inch. Below nipple level no movement on ordinary inspiration, 1 inch expansion on deep inspiration. Weight on admission, 8st. 9lbs., on leaving Hospital, 9st. 2lbs.

Unfortunately, I did not, in this case, *measure* the mobility of the chest on admission. But my observation of "expansion with inspiration impaired" refers to total mobility of chest and the circumferential movements of the chest at that date may be taken as those of a chest fixed by the emphysematous condition of the lungs. The later measurements, however, which were similarly taken in the position of repose are almost identically the same; but at this period the mobility of the chest, its capability not only of expansion but also of contraction, was nearly equal to that of health, and bore valuable testimony to the recovery of the lungs in vital capa-

city, a point not to be estimated by reference to chest expansion alone.

Again, it may be observed in this case as in many others, that mere rest from his dusty occupation did not suffice for his recovery; although doubtless had he remained in pure air a sufficient length of time, he might have recovered without treatment. His attacks had the paroxysmal character peculiar to asthma, coming on towards the small hours of the morning, *i.e.*, after a certain period of repose whilst the breathlessness and cough of the bronchitis and attendant emphysema were constant through the day. The effect of the stramonium and iodide of potassium mixture was very striking and immediate. Having regard to the period at which the dyspnœa became distressing, the mixture was so ordered that by midnight he had taken in the course of twelve hours a grain of stramonium extract, and twelve grains of the iodide. To which of these two drugs—both active in the relief of asthma, the amelioration of symptoms was due I am unable confidently to say. The mixture was not prescribed experimentally, but as a well-tried and trusted remedy in similar cases.

The mechanism of the dyspnœa, however, was pretty obvious; the man had an unduly secreting, and probably a somewhat thickened bronchial tract, with great irritability of the bronchial muscular apparatus, and constant tendency to spasm of the tubes. At a certain time after repose, secretion would accumulate and give rise to spasm. The hypersensitivity upon which the bronchial spasm depended was at once lessened by the stramonium, whilst the iodide had a more permanently alterative action upon the mucous membrane and its secretion. This explanation would be perhaps generally accepted, but one has abundant experience with regard to iodide of potassium, that it alone frequently suffices very rapidly to relieve asthma. In what way the drug does this is, so far as I know, entirely unexplained. The patient left

the Hospital well, but after a time, failing to find other work, was reduced to the necessity of recommencing his old employment, and soon had a return of all his old symptoms for which he was treated elsewhere by ordinary remedies (ether and expectorants) without avail.

In the trade of fret-cutting the operator has constantly, with his mouth, to blow away the fine wood dust that collects upon his work, and thus necessarily inhales much of the dust. The patient above referred to, stated that he could distinguish by the taste the different kinds of wood, and he found rosewood (the taste of which he compared to cayenne pepper) to be the most irritating. Walnut wood was more astringent and bitter but less irritating.*

* Whilst these proofs are passing through the press I have again seen this patient in fairly good health, having for some time abandoned his former work. There does not seem to be any reason, however, why a respirator perforated with a tube, should not be so adapted as to enable the man to resume his occupation without injuring his health, and I have had such an apparatus constructed for him.

CHAPTER II.

REMARKS ON PULMONARY VESICULAR EMPHYSEMA.

DEFINITION—Nature of defect in respiratory mechanism in emphysema—

Conditions present in emphysema recounted, with comments—Etiology of the disease, partly mechanical; observations of Jenner, Gairdner, Rindfleisch and Greenhow referred to. Atrophic changes, precede and accompany the dilatation: earliest stage accounted for. Illustrative case of emphysema. Remarks on treatment.

THE emphysema here referred to is the hypertrophous or large-lunged emphysema so named by Sir William Jenner, and may be defined as a dilatation of the air-cells of the lungs with antecedent or associated atrophic changes. Atrophous or senile emphysema scarcely merits consideration as a separate disease. The interlobular emphysema of Laennec is rarely met with save as the result of accident or *post-mortem* decomposition.

As hinted in the remarks upon the condition of emphysema present in the case briefly recorded in the last chapter, the perfectness of the function of respiration consists quite as much in the power of contracting as in that of filling the chest. It is this power of contracting the chest that is lost in emphysema. The lungs in this disease have lost their reserve elasticity, they no longer tend further to contract at the completion of expiration: nay, expiration is never completed, the thoracic parietes and diaphragm instead of being drawn inwards by the traction of the lungs recoil simply to their position of repose, and oppose their dead weight to the inspiratory muscles instead of aiding the action of these muscles by their elastic rebound. Hence in emphysema as I have elsewhere endeavoured to show⁴ the inspiratory act commencing when healthy calm inspiration ends, has to overcome, (1) what remains of the elastic resistance of the lungs,

⁴ *Medico-Chir. Trans.*—Vol. lix, p. 169.

(2) the inertia of the parietes, and (3) the elastic resistance of the parietes, instead of, as in health, having to deal with the elastic resistance of the lungs alone, and being in this work aided by the outward spring of the ribs. Consequently, in marked cases of emphysema, the breathing is always forced and more or less dyspnœic. Let me now briefly recount the conditions present in emphysema, making such additional comments as seem called for.

(1). The lungs are permanently expanded to about the position of ordinary inspiration, their elasticity being, so to speak, relaxed to this point. The individual air-cells are correspondingly enlarged and the pulmonary vascular system lengthened.

The enlargement of the air-cells commences as pointed out by Rindfleisch⁵ in the central infundibular cavity which, normally about one-third larger than the alveoli opening into them, become proportionately much larger, the septa of the alveoli meanwhile becoming shrivelled and atrophied to mere ridges or lines.

(2). The texture of the lung is impaired more or less.

(a). In cases of acute emphysema, as after whooping cough or acute bronchitis, the impairment is slight and remediable.

(b). In cases of long-standing emphysema, due to repeated attacks of bronchitis or asthma, the nutrition of the lung suffers, many of the small vessels of the pulmonary system become obliterated, the alveoli of the infundibula atrophy and sometimes coalesce, and the stroma of the lung becomes granular, degenerated, and somewhat thickened.

(c). In cases of hereditary emphysema the atrophic changes are primary and are more marked.

The opinions expressed by writers on the minute anatomy of emphysema are very guarded, and more complete observations are much wanted. No doubt in acute emphysema loss of tone from repeated over-distention, e.g., during par-

⁵ *Pathological Histology*. Syd. Soc. Edition.—Vol. ii, p. 7.

oxysms of coughing, is the chief and remediable defect present. In chronic cases of constitutional or hereditary origin, however, the atrophic changes originate in fatty degeneration of the epithelium and vessels of the lung. In cases again in which the emphysema has succeeded to oft-recurrent catarrhs, ill-developed fibrous or fibro-cellular tissue, the result of repeated and long-continued congestions, toughens the lung-texture and leads to its subsequent degeneration.

Whatever may be the exact pathological processes leading to emphysema, the chief results are loss of elasticity of lung and obliteration of vessels, the latter result being in part due to primary atrophic changes in the capillaries extending back to larger vessels, in part to secondary atrophic changes from narrowing of the blood-current as the vessels become lengthened, thus causing them to wither from deprivation of proper blood-supply.

(3). In consequence of the relaxed elasticity of the enlarged lungs they no longer exercise any traction upon the mediastinum except during inspiration. Hence an important, because constant, aid to the return of blood to the heart is lost.

It is commonly held that the large lungs in emphysema are, so to speak, pent up in the chest, and exercise pressure upon the heart between them and upon the ribs and diaphragm which enclose them. A little reflection and clinical observation will, however, render it clear to anyone that this supposed pressure of the lungs upon the surrounding parts is, if not impossible, of infinitely rare occurrence and minute degree. The enlarged thorax, flattened diaphragm, and lowered heart, are all phenomena due to defective recoil of the lungs, not to their forcible distension: they are to be observed in health on the lungs being inflated during deep inspiration, which position they retain in emphysema.

(4). In addition to the negative impediment to the circulation referred to in proposition 3 there is a positive impediment in the stretched, and in part obliterated capillaries of the lungs.

(5). In consequence of the two last-mentioned conditions the venous system is over-full and the over-worked right heart thickens. The increased power of right ventricle is not however adequate to contend against the ever present and from time to time (during bronchitis or asthma) increased resistance to the pulmonary circulation. The whole venous system becomes engorged, and especially the hepatic and portal systems : the circulation is carried on at a heightened pressure, venous haemorrhages occur, and oedema, commencing at the legs, sooner or later sets in.

When we come to inquire how this over-expansion of the air-vessels is produced, we are met by various explanations, none of which alone is sufficient to explain all cases. Sir William Jenner has shewn that expiratory effort,— during straining or coughing, particularly the latter, is an efficient cause of general emphysema, those portions of lung which are least supported, viz., the apices and anterior margins and also the parts corresponding with the comparatively yielding intercostal spaces, becoming first affected. But with the production of emphysema in these portions of the lungs a shifting of the relationship between the lung and the thoracic surface takes place, and parts which were originally in apposition with the ribs come to be opposed to inter-spaces, and in their turn yield before the distending force of air pent up and compressed during cough.⁶ Again, in the production of local emphysema inspiratory pressure is undoubtedly an important agent. Thus, if a man gets one lung disabled or bound down by some inflammatory process (as old pleurisy or chronic pneumonia) the other lung perforce be-

⁶ Sir William Jenner (Reynolds' *Syst. of Med.*—Vol. iii, p. 478) refers to Mendelssohn as having in a paper "*Der Mechanismus der Respiration und Circulation*" in 1845 anticipated him in this view respecting emphysema. The authorship and advocacy of the view in this country rests, however, with Sir W. Jenner.

comes more capacious; whether this extra capacity shall be derived from true hypertrophy or mere dilatation (emphysema) depends upon the nutritive vigour of the patient. During bronchitis certain of the air-tubes may become occluded by mucus, and the inspiratory force then operates as a distending power upon the remaining portions of the lung until the deficiency in air-space is compensated for. Professor Rindfleisch, who adopts Dr. Gairdner's view in regard to the production of emphysema, further holds that "during the antecedent bronchitis, first one then another bronchial tube is plugged with secretion, and so first one then another segment of lung is subjected to an abnormal degree of (inspiratory) distension."

In the histories of the majority of cases of pulmonary emphysema, however, facts are wanting which would justify our accepting the *inspiratory* cause as effective in their production. Indeed both the expiratory and inspiratory theories imply the presence of some pre-existing bronchitis, or some local lesion disabling a portion of the lung, or the pursuance of some occupations requiring repeated efforts, during which the glottis is closed and the chest compressed, all of which are wanting in a considerable proportion of cases. In truth the emphysema frequently precedes the bronchial affection, although it is subsequently aggravated by the first attack of bronchitis. When emphysema does succeed to repeated attacks of bronchitis, as one must readily admit is very commonly the case, the tussive expiration is a much more powerful cause of over-distension of lung than any inspiratory efforts, (short of that prolonged and urgent dyspnœa, which is not met with in these cases) could be.

Lastly, if we admit as I think we must admit, with Dr. Greenhow and others, that a failure of nutrition is in a large proportion of cases the predisposing cause of emphysema, we need go but little further to explain the occurrence of that disease in its earlier grades. The effect of damaged textural

nutrition of the lungs is to relax them by impairing their elasticity, and, as I have elsewhere shewn, the thoracic elasticity or resilience tends to expand the chest to the degree of from one to four millimeters in each direction. As the lungs relax their elasticity, they yield to this traction exercised upon them by the thoracic wall, and to the weight of the abdominal organs dragging upon the diaphragm, and thus we get expansion of lung to a degree any extension of which will suffice to cause marked dyspnoëic symptoms. Impairment of a degenerative kind in the elasticity of the ribs and cartilages, referred to by Freund, may take place *pari passu* with, or may even precede, this change, but this increased rigidity of cartilages would not operate in any other way than by increasing and rendering more fixed the enlargement of the chest.

It is from this view of the mechanism of emphysema that one can appreciate with increased force the fact that it is in the lessened power of contracting the chest and emptying the lungs that the great defect lies in emphysema. The act of expiration is never completed, there is too much residual air constantly in the lungs, and inspiration is short and jerking from the act being commenced where it should end.

The physical signs of emphysema will be sufficiently indicated in the description of the following extreme case of the disease.

George D—, oil and tallow warehouseman, admitted into the hospital under my care in December, 1875. The patient was a tall man with no hereditary tendency to phthisis, but of gouty parentage on the father's side. He had up to three years ago never been laid by with any illness. At that time he had an attack of bronchitis and since then had complained of shortness of breath and cough, constant in the winter, attended with frothy and viscid expectoration and with a sense of constriction felt below the ribs. He had never spat blood nor suffered from hectic; lost and gained flesh rapidly.

His principal symptoms on admission were, great shortness

and difficulty of breathing; the head and face becoming congested, almost cyanosed at times, during attacks of dyspnoea and cough. No pain, but great sense of constriction below the ribs. Appetite bad, digestion tolerably good, bowels irregular at present relaxed by medicine. Sleep fairly good.

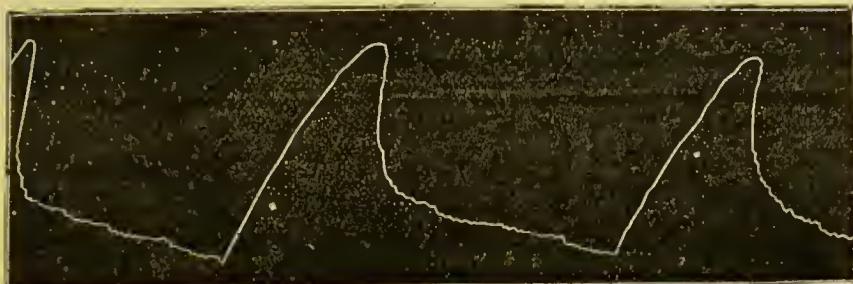
Physical signs.—Pulse 86. Respiration slow and forced. Temperature normal. Great oedema of lower extremities and scrotum. Chest greatly expanded: extraordinary muscles of respiration prominently employed, respiratory movements mainly thoracic. Intercostal spaces above nipple level slightly depressed during inspiration, becoming quite level with the ribs on expiration. Below nipple level intercostals greatly depressed during inspiration, becoming level with ribs or even slightly puffed outwards during expiration, seventh and eighth ribs yield inwards with inspiration. Heart's impulse most perceptible at left costal margin level of tip of ensiform cartilage.

Girth of chest on each side above nipple level 18 inches. At level of base of ensiform cartilage right side, $18\frac{5}{8}$, left $18\frac{1}{2}$ inches, $\frac{1}{4}$ inch extreme movement with inspiration in both these situations. The whole chest resonant both anteriorly and posteriorly down to the margin of the ribs both in front and behind; at posterior bases on both sides fine bubbling râles heard principally with inspiration. Similar râles heard over lower two-thirds of the right and left fronts. Apices clear, cardiac sounds unattended with bruit. Abdomen somewhat distended, contains a small quantity of fluid. Liver depressed.

The subjoined tracings show very well the nature of the respiratory movements in this case, they were taken by a very simple apparatus consisting of a straight rod connected by a flexible joint with an expanded button to apply to the chest, the other end scratching upon a horizontal sphygmograph plate previously smoked.

Tracing A, represents the movement of the sternum at the level of the third cartilage. (The man was sitting in a chair

FIG. 8. (A).



Tracing of respiratory movement showing total forward thrust at third mid-sternum.

FIG. 9. (B).



Tracing, showing recession c. c. c. during inspiration, seventh rib, axillary line.

with his back resting against a flat board). It is equivalent to exaggerated thoracic breathing although the patient was inspiring in the degree natural and necessary for him. Tracing FIG. 9 was taken at the 7th rib in the lateral region right side, and shows a distinct recession *c. c. c.* during each inspiration.⁷ This was perhaps due to the bases of the lungs being in some measure disabled by secretion, but this disablement and collection of secretion was undoubtedly in the greatest measure owing to the practical paralysis of the diaphragm in consequence of the flattening of its arch. The thoracic movement was certainly somewhat in excess in this case from the same cause, but in most cases of emphysema the respiration becomes thoracic rather than abdominal.

The patient died after he had been in the hospital three weeks from general dropsy and cyanosis.

The *post-mortem* examination revealed the usual phenomena of large dilated heart, the right side most affected, the tricuspid

⁷ The right lung was found *post-mortem* to be more highly emphysematous than the left.

orifice measuring $6\frac{1}{2}$ inches in circumference. Large emphysematous lungs, the emphysema most marked at the anterior and upper parts and in the right lung, there being oedema and slight congestion at both bases. There were no signs of active bronchitis, the bronchial tubes contained a frothy, thin fluid. Spleen hardened, kidneys mechanically congested, liver enlarged and fatty.

With regard to the *treatment* of emphysema it is well to remember that all the functions are, in this disease, carried on under an excessive arterial pressure, due to obstruction on the venous side in consequence of a narrowed passage through the lungs. Whilst the right ventricle remains vigorous it can by increased toil reduce to a minimum the backward congestion; when it fails we begin to have symptoms referable first to the digestive organs: habitually slow and impaired digestion, with a disposition to haemorrhoids; hepatic congestions and attacks of gastric catarrh bearing witness to difficult circulation in the portal system. If the patient has an attack of bronchitis the increased stress in the lungs tells back still further, and we begin to get oedema of the legs from retarded flow through the systemic capillaries. This oedema may for a time clear off with the subsidence of the bronchitis, but, sooner or later, it becomes permanent and extends more generally through the cellular tissue and into the serous cavities. A restricted well-assorted diet and the relief of the circulation by increased action of kidneys and bowels are the general measures of treatment suggested by these conditions. We can only, so far as we at present know, treat the lungs by carefully avoiding fresh catarrhs, and by promptly attending to such when they occur.

The true value of the employment of compressed or rarefied air in the treatment of emphysema, has not yet been fully ascertained. But in endeavouring so far as is possible, to form a judgment upon the subject, we must carefully distinguish between condensed or rarefied air climates or baths, and the

same modifications of air pressure brought to bear upon the interior of the lungs only, not upon the whole body. If a man, himself surrounded by the ordinary atmosphere, inhales from a compressed air chamber, his lungs become forcibly expanded by an air rich in oxygen on account of its concentration. Such a treatment may be useful in certain cases of collapse, from long continued compression of the lung by fluid or from inflammatory thickening, but it is not adapted for a patient whose chest is already over expanded⁸ from impaired power of expiration. The effect too of inhaling compressed air in this way is to render the lungs more anæmic, whereas, in emphysema, they are already defectively supplied with blood; and the only advantage, on the other hand, to be gained is the doubtful one of displacing the stagnant residual air by one highly charged with oxygen.

If, however, the emphysematous patient be immersed in an atmosphere compressed, say, to half again the density of ordinary air, his respiratory mechanism is neither helped nor impeded by the new conditions, for the atmospheric pressure is not only increased on the inner surface of the lungs, but also, and similarly, over the whole body. He is simply breathing a concentrated air, but as the vital capacity of his lungs is considerably diminished, some advantage may be thus obtained from the air inhaled being (bulk for bulk) richer in oxygen. Be it remembered, however, that with increased density we get diminished *mobility* of the air particles, so that the osmotic interchange of gases on which respiration truly depends is more slowly effected. Again, the heart's action is somewhat impeded by the increased pressure upon the vessels: still it is true that emphysematous people do well at the sea level, and some derive benefit from the use of compressed air baths, although these baths cannot in any sense be regarded as curative in emphysema.⁸

⁸ *Vide Walshe, Diseases of the Lungs*, 4th edit., p. 333. *Braun on The Curative Effects of Baths and Waters*, edited by H. Weber, M.D.,

It is generally held that elevated climates are unsuitable for patients with emphysema, inasmuch as such patients must breathe more deeply to obtain the same amount of oxygen from a rarefied atmosphere. Respiration in a rarefied air would tend thus rather to increase the evils already present in an over expanded chest with deficient power of expiration. But there are some considerations from which it would appear that such an atmosphere is much less unsuitable to emphysematous patients than one might at first assume.

(a.) In the first place it must be remembered that with an abundant air supply,⁹ we use only a very small proportion of the oxygen present in the air for our respiratory purposes, in other words expired air is not nearly exhausted of its oxygen; (b.) secondly, although the air of elevated regions is rarefied, its particles are more actively mobile, and oxygenation is relatively quickened. Thus Tyndall and Frankland¹⁰ have shown that the loss of weight of a candle burning on Mont Blanc at an elevation of 12000 feet, is identically the same as that of another candle of similar dimensions, burning in the valley of Chamouni below; (c.) thirdly, the circulation through the lungs, as elsewhere, is carried on at less pressure in elevated regions, and the heart, tuned originally to lower latitudes, finds relief in this way.

There are many other features, besides the mere elevation with which we are now only concerned, which would of course have to be taken into consideration in coming to a decision as

p. 53. Cohen on *Inhalation in the Treatment of Disease*, Philadelphia, 2nd edit., p. 41. The simplest compressed air bath consists of an iron chamber, into which air is gradually pumped to an excess of half an atmosphere or more. The bath is in daily use at the Ben Rhydding Sanatorium.

⁹ It has been proposed to use rarefied air baths, but I need scarcely say that the above remarks refer exclusively to the rarefied air of elevated regions, inasmuch as one would hardly with seriousness suggest placing any patients under an air pump!

¹⁰ Quoted by Braun, *loc. cit.*, p. 59.

to the relative advantages of high or low climates in the treatment of emphysema. No statistics upon the subject have, so far as I know, been published.

But inasmuch as it is the *expiration* which is at fault in emphysema, and since, from the defective lung recoil, there is an undue amount of residual air retained in the lungs, the most decided treatment, indeed the only real treatment of the physical kind under consideration, is that of causing the patient to expire into a partially exhausted chamber. This method of treatment has been tried by several good observers, especially by Waldenburg, Schnitzler¹¹ and Berkart. Dr. Waldenburg¹² relates several cases in which marked benefit has ensued upon the employment of this treatment; and although most of his results are complicated by the simultaneous use of turpentine, saline spray or other inhalations, yet the relief of symptoms and gain in vital capacity cannot by these other means be accounted for. In most of his cases Dr. Waldenburg caused his patient to inspire compressed air and to expire into rarefied air.¹³ Dr. Berkart¹⁴ has also devised an ingenious little instrument by which a chamber communicating by a tube with a suitable mouth-piece provided with inspiratory and expiratory valves, can, at the moment of completion of the inspiration, be exhausted, and thus remove more residual air from the lungs.

These mechanical methods of treatment are attended with

¹¹ *Wiener Klinik*, 1875, vol. 6. See abstract of Dr. Schnitzler's views in Dr. Dobell's Reports, vol. ii, p. 114.

¹² *Die Pneumatische Behandlung der Respirations und Circulations-Krankheiten*. Berlin, 1875, pp. 385—411.

¹³ Waldenburg's apparatus is an adaptation of Hutchinson's spirometer with weights so adjusted as to exercise positive or negative pressure upon the contained air as may be required. It is described and figured in his work (p. 128) and also in Cohen's work (p. 45).

¹⁴ *Lancet*, Nov. 25th, 1871.

many practical difficulties, and the results obtained are only as yet sufficiently encouraging to warrant further investigation. And further, it must always be carefully remembered, that we have in emphysema invariably to deal with *a more or less advanced degenerative disease of the lung texture.*

In regard to both prognosis and treatment, however, the heart is the real key to the conditions present in emphysema, and by maintaining the power and nutrition of the heart, and especially of the right ventricle, life and comparative health may be preserved for years. The regulation of exercise taken is of the utmost importance, for on the one hand, if the patient give up exercise altogether the heart becomes languid, its muscular substance flabby and deficient in reserve energy. On the other hand, any active exercise such as decidedly to hurry the respirations, causes too high a degree of pressure upon the right side of the heart and may lead to rapid dilatation and consequent dropsical symptoms. Absolute rest should be insisted upon during any intercurrent bronchial attack, and in the intervals of comparative health, steady walking exercise on level ground is of equal importance. From time to time, and especially during convalescence from bronchial attacks, digitalis is most valuable in doses sufficient to steady and give tone to the heart. In these cases it usually acts also as a most efficient diuretic. It may be combined with expectorants or with iron or bitter tonics as the case may require. A mistake, however, which is commonly made in the treatment of these attacks, is the too long continuance of stimulating expectorants. The stethoscope is doubtless concerned in this error, for bronchial sibilus and mucous râle persist long beyond the period when the drugs they are supposed to indicate cease to be of use. Indeed it is not uncommon in emphysema for there to be some loose crepitant râle permanently present over the posterior bases.

Occasional mercurial aperients, and the more frequent use of

the natural aperient waters (especially the Hunjadi water), serve to maintain equilibrium in the portal system, and the digestive functions may be further aided and supplemented by the administration of pepsine. For those who can afford it, a seasonable change to one of the Mediterranean health resorts, or to Rome or Egypt, is highly to be recommended. But in our own country at Torquay, St. Leonard's and Bournemouth, Brighton, Ramsgate, &c. can be found climates sufficiently sheltered for cases of this kind. Indeed many patients do much more wisely to remain at home and devote some extra outlay to the better arrangement of their own homes for winter residence.

CHAPTER III.

PNEUMONIA.

PNEUMONIA—a disease more familiar than common. Sketch of general symptoms of a case in mid-career: more detailed symptoms and signs.—Anatomical characters—Promontory symptoms, various in character and may suggest other affections than pneumonia—Signs of pneumonia continued. Defervescence. Resolution: occasional occurrence of secondary fever: explanation of, including *r  sum  * of morbid and restorative processes in pneumonia—Etiology—Definition of pneumonia—Prognosis and Treatment. Excessive temperature, abscess, gangrene and empyema, the chief dangerous events that may occur in pneumonia: their treatment.

THERE is perhaps no disease with which students are more familiar than pneumonia in its acute and typical form. It is one of the standard diseases for clinical demonstration in our hospitals. Hence it is not necessary in this place to do much more than recapitulate its principal features and to add some remarks of a practical kind regarding the diagnosis, treatment, and the future course of the disease.

The general aspect and symptoms of pneumonia are usually such as to leave but little room for doubt as to the nature of the case. The flushed look and burning skin, the hurried noiseless breathing, and rapid but regular pulse, the frequent short cough half-stifled from pain, the dryish thickly-coated tongue, and the singular prostration of the patient are a group of signs which, supervening speedily upon a well-marked attack of shivering, cannot be otherwise interpreted than as being those characteristic of an attack of ordinary *acute basic pneumonia* in full intensity.

On closer examination the respirations are found to number about 40 in the minute: they are not obstructed nor attended with marked action of the nares. The pulse 120;

the temperature 104° . On inspecting the chest its movements are found to be chiefly one-sided, but there is no apparent difference in size on the two sides, and the heart is found beating in its normal position. The movements of the affected side are voluntarily restrained by the patient, and a severe pain referred to this part often cuts short the cough or any attempt to draw a full breath. The percussion signs in front are not materially altered; on auscultation the respiratory murmur is found here to be weakened on the affected, exaggerated on the healthy side. Posteriorly, over the base of the affected side the percussion note is dull, but not without some wooden quality of tone quite distinct from the dead flat note of effusion. The dulness extends upwards to a variable height and over the dull portion the respiration is characteristically bronchial or tubular, the voice sounds well conducted, broncho-phonic, and, especially towards the upper limits of dulness, the peculiarly explosive fine inspiratory crepitation of pneumonia is heard. If there be any expectoration it is scanty, viscid, frothy, and more or less rust-coloured from blood staining. The urine is scanty and concentrated, deficient in chlorides and perhaps yields a thin cloud of albumen.

If, as rarely happens, the patient should die at this stage, we find on inspection the affected portion of lung bulky, heavy, and solid to the feel. The pleural surface is covered with a thin layer of soft fine granular lymph which can be readily scraped off, exposing the glistening pleura beneath. On section the lung is firm and dry, presenting a red granular surface which is readily broken by the pressure of the finger. There may be a little frothy secretion in the bronchial tubes, the mucous membrane of which is injected. A portion of the consolidated tissue sinks at once in water. These characters are distinctive of the stage of red hepatization, the *second stage* of pneumonia. And, on making a section at the upper limit of the consolidation, there may possibly

still be seen the engorgement or hyperæmia with serous frothy exudation, characteristic of the *first stage* of the disease.

We have for convenience of description taken the symptoms and signs of pneumonia at its most striking period. It is at this period (the second or third day after the rigor), that we are most commonly called upon to see a case of pneumonia, but the chest symptoms are the same from the first onset of the attack, and the physical signs vary chiefly in the increasing intensity of the bronchial breathing, and the gradual extinction of the crepitus as the consolidation marked by percussion dulness extends from below upwards.

There is no doubt that the general symptoms of pneumonia may precede the appearance of physical signs by a very perceptible interval of time during which, on examining the chest, no dulness is as yet to be found. The breath-sound at one base has, however, a peculiar rough, harsh quality very like that of exaggerated breathing and slight crepitus may be audible. Soon, in a few hours, the characteristic pneumonic crepitus is abundantly evident, whilst the percussion note, although shortened and heightened in pitch, is, as yet, by no means dull. In other cases again the physical signs of complete consolidation may develope so rapidly as to render no previous stage noticeable. And here it is well to remark that there may be observed in different cases some diversity in the general symptoms which precede the pulmonary lesion. The rigors may be strongly marked and speedily followed by severe headache, and even somewhat violent delirium such as to suggest meningeal inflammation. But the hurried *regular* breathing must not escape notice. In other cases a marked icteric tinge of skin with gastric disturbance suggests the oncoming of jaundice, but the fever runs too high for simple jaundice and the breathing and pulse also betoken a more acute disease. Finally, the local symptoms, severe pain and dyspnoea, may be those which chiefly attract attention, and these are most commonly cases of the worst augury as re-

gards the lungs—cases in which the disease is in the smallest sense idiopathic and has rather been, so to speak, forced upon the patient by exposure to cold.

To follow our assumed case of pneumonia further onwards:—the temperature rising after the onset with rigor to 104° hovers thereabouts with but little variation for four, five, six or seven days and then rapidly falls within forty-eight hours to normal. The patient who has been suffering throughout from complete anorexia, thirst, restlessness, and increasing weakness, with dyspnoea, troublesome cough, and blood-stained expectoration, is now rapidly relieved from many of these symptoms. The sense of dyspnoea is greatly lessened although the breathing is still double-quick. The pulse becomes quiet, the skin moist, and the tongue begins to clean at the tip and edges. Appetite does not yet return but the sleep is quiet and refreshing.

On examination, however, the physical signs will be found to have but little changed, dulness and bronchial breathing being as distinct as ever. At the upper boundaries of the consolidation, however, the crepitation if present, or, when it returns (for it often is but little marked or disappears for a few days) is found to have altered in character. It is larger, moister, less explosive, and is heard during expiration as well as inspiration although still most abundantly with inspiration. This *sub-crepitant râle* marks the commencing resolution of the hepatized lung and extends downwards as the consolidation slowly melts away. The sputa now become more or less opaque although usually still scanty. In some cases the expectoration at this stage is muco-purulent amounting to several ounces during the day. But it yields no lung tissue to microscopic examination after boiling in caustic soda solution. Sometimes again from first to last there is no expectoration in pneumonia. At this stage of the illness there is in some cases a slight return of fever but of a different type to that of the original disease. The temperature assumes a hectic type with a moderate daily

rise to 101° or even 102° , and in these cases slight daily chills are complained of. These symptoms are somewhat alarming, and suggest the possibility of some caseation or softening of lung-tissue proceeding, or of purulent effusion into the pleura. But they do not either of them necessarily bear such untoward meanings. I have observed such symptoms now in three cases in all of which the pulmonary lesions entirely cleared up. Their *rationale* is clear and it is somewhat surprising that they are not more often observed and have not, so far as I am aware, been described.⁸

If a portion of a lung which is becoming again crepitant be examined it will appear to be infiltrated with pus, and the term "purulent infiltration" has indeed been used to describe the condition present in this stage of grey hepatization. The texture of the lung is, however, not infiltrated, the "purulent" stuff may be washed away from the air-cells leaving them intact (Jürgensen): in brief, the first stage of pneumonia, anatomically speaking, consists of an active hyperæmia of the lung (active congestion), which, in the second stage (red hepatization), results in the effusion of fibrinous lymph into the air-cells, coagulating there so as to occupy them with leucocytes entangled in the fibrinous meshes; in the third stage (grey hepatization), the effused lymph becomes opaque from fatty transformation of its fibrinous portions, and this proceeding to liquefaction, the entangled corpuscles, themselves also granular, are set free and the fatty emulsion thus produced is ripe for absorption or expectoration. It is, however, probable that more leucocytes do escape into the air-cells at this latter stage when the minute vessels are relieved from pressure. The expectoration in

⁸ Dr. Parkes observes, however, "the exudation * * * may contaminate the blood during softening to such an extent as to lead to renewal or increase of the fever and inflammation of other parts; or to coagulation of the blood in the heart or great vessels."—Clinical Lecture on a Case of Acute Pneumonia.—*Medical Times and Gazette*.—Feb. 1860.

this stage of pneumonia becomes opaque and more or less purulent, but as a rule it continues to be very scanty, and it is perfectly clear that in most, if not in all cases, the great bulk of the morbid products effused into the lung, having become emulsified by fatty degeneration, is again absorbed. It is during this period of reabsorption of inflammatory products that we are apt to get the symptoms of hectic more or less decided. It takes a considerable time—from three to six weeks, or even longer for the inflammatory products of pneumonia to become entirely absorbed and for the lung to recover its function.

Although as a rule the consolidation of pneumonia clears up pretty uniformly in the inverse order of its formation it certainly does not do so in all cases, and detached islets of resolving exudation sometimes give rise to physical signs—largish clicks and circumscribed blowing sounds, which it may be almost impossible to distinguish from those of pulmonary disintegration. A careful examination of the sputa in the manner already explained will help us much to a right view of such cases.

The etiology of pneumonia calls for a few remarks. The disease present is rarely if ever a simple inflammation of the lung and most observers agree in this that, whatever may be the exciting cause of pneumonia, there is a morbid general condition behind upon which the peculiar lesion elicited is dependent. *Pneumonia may indeed be defined as an acute, non-contagious febrile disease, accompanied by inflammatory exudative consolidation of the lung.*

Exposure to cold is the chief reputed cause of pneumonia, and the prevalence of north and north-east winds, especially when supervening upon mild moist weather, is a fruitful source of the disease. But this same exposure to cold is the "cause" of much more than half the ailments of mankind.

It is to be noted that pneumonia occurs most frequently in the vigorous period of adult life, and although it is said to attack

by preference those who are constitutionally feeble or diseased, yet persons of previously sound health are by no means secure from the malady. There is indeed good ground in experience for the belief that typical pneumonia is much more commonly met with in persons of sound constitution, but who have had their general health lowered by some temporary cause, such as over-anxiety, over-work, prolonged debauch, or other depressing influences. The pneumonias to which cachetic (scrophulous or tubercular or syphilitic) persons, or those actually suffering from renal or cardiac disease are liable, are broncho- or catarrhal pneumonias, or are local inflammations determined by mechanical congestion, as in the congestive pneumonia of low fevers or cardiac disease, or secondary to haemorrhage into the lung. The morbid constitutional states in which true pneumonia is most apt to occur would seem to be the rheumatic, the gouty, and the alcoholic. Let me repeat, however, that true croupous pneumonia most commonly arises as quite a primary and independent disease.

Prognosis and treatment.—Patients with acute pneumonia as a rule recover, but the disease is one in all cases attended with danger. The quarters whence danger is apt to appear are—1, from excessive fever and consequent cardiac exhaustion (Jürgensen); 2, from involvement of the second lung; 3, from intensity of the inflammatory process, or from cachexia on the part of the patient, leading to abscess or gangrene of the lung.

The *treatment* of a case of pneumonia consists first of all, and most importantly, in watchful nursing. The room should be kept at a temperature of 65° , well ventilated, with the air slightly moistened. Hot poultices constantly kept applied over the affected part of the chest give great relief to pain and mitigate the intensity of the local inflammatory process. With old people and children the poultices are sometimes found to be oppressive, and will then be better exchanged for a thick layer of cotton-wool covered with oil-silk. If the pain

be severe the application of a few leeches may be suggested. In other cases some mustard is usefully added to the first poultice or a blister applied under the poultice.

Simple salines are at first indicated, and milk and seltzer or barley or rice water may be freely allowed with nutritious slop diet. In cases in which there is much adynamia more stimulating food such as Brand's beef-jelly, cream and brandy, &c., must be allowed in carefully regulated quantities. As soon as the fever subsides the salines should be given less frequently, and a small dose of mineral acid administered twice daily, the diet being gradually improved as the tongue cleans. Cotton quilting or spongio-piline applications should now be substituted for the poultices, and slight counter-irritation kept up by occasional mustard poultices or other stimulating applications. Finally, change of air is usually requisite successfully to get rid of the last remnants of the local disease. Such is a sketch of what is the most successful routine treatment of ordinary cases of croupous pneumonia. And whether the pneumonia affect the base or the apex of the lung, the treatment is the same; in the latter case, however, due regard being given to the, as a rule, less sthenic nature of the disease.

But in cases of pneumonia danger may at any time set in and require more energetic treatment. *Excessive fever, involvement of the second lung, and the occurrence of abscess or gangrene* being as before said the especially dangerous incidents of this disease.

The German physicians are strong in their advocacy of the employment of cold locally or generally in the treatment of pneumonia. But statistics certainly do not prove their treatment of pneumonia by cold baths to be successful⁹ and cases in this country are of very rare occurrence in which

⁹ Compare the tables quoted in Jürgensen's article on *Pneumonia* in Ziemssen's *Cyclopædia*, Vol. V., and Dr. Sturges' work on *Pneumonia*, Appendix G. giving the results of different treatments in the tabular form.

physicians would advise its adoption. The fever of pneumonia, although sharp, is of short and tolerably definite duration, and it does not as a rule call for any decided interference. In some rare cases, however, in which the temperature ranges very high (105°) and nervous and cardiac exhaustion threaten, treatment by cold is not only justifiable but imperative. In these cases delirium is usually a marked symptom and it is probable that iced applications to the head, and more especially the convenient ice-water cap recommended by Mr. Knowsley Thornton¹⁰ for the purpose of reducing temperature in excessive fever following ovariotomy, would give great relief. These cold applications to the head seem to have an effect more immediate than is explained by the mere withdrawal of heat, probably influencing in some way the nervous-centres upon which depends the excessive production of heat. Tepid sponging may be employed, or the warm bath ($80^{\circ}\text{F}.$) or wet pack may be had recourse to. By some such means the temperature *must immediately be lowered* when it mounts up beyond the limits normal to the disease. Stimulants must at the same time be administered to any amount needful to maintain the heart's action, and strong beef-tea, milk and cream, must be liberally supplied in frequent small quantities. Cases of pneumonia certainly as a rule resolve better with the aid of but little or no stimulants, but in the exceptional cases now referred to they must be administered with a bold but careful hand. I have heard an able physician, of too old a school to recognise hyper-pyrexia as an entity, boast that with the help of the wine-bottle he has never lost a case of pneumonia.

The second lung sometimes becomes early involved; in severe cases one must, however, in listening over the healthy base, be careful not to mistake sounds as being generated there which are really conducted from the other side, a mistake very excusable and probably often made. More

¹⁰ *Med. Chir. Trans.*, Vol. ix., p. 301.

frequently the second lung is affected by a distinct attack with an exacerbation of fever when the lung first affected is about to resolve. Cases of double pneumonia frequently do well. They are dangerous, however, in proportion to the amount of lung simultaneously disabled. In such cases small bleedings may theoretically be indicated, but are in practice very rarely to be adopted.

In cachectic cases, those of persons whose constitutional powers are prostrated by intemperance or syphilis, an abscess frequently forms in the inflamed lung. This occurrence is marked by chills and sweats, and by the expectoration by and by of a large quantity of purulent matter, sometimes already foetid, always becoming so in a few hours. Ammonia and bark, or quinine and mineral acid, with abundant support are at once indicated on these symptoms presenting themselves. These cases also, perhaps more frequently than not, do well, but they require prolonged treatment and change to seaside air. Unfavourable cases proceed to breaking up of other portions of the lung and to chronic or sub-acute phthisis.

Cases, also most common amongst drunkards, but occasionally seen in other persons of somewhat advanced age, or much depressed in health, in which a gangrenous taint is noticeable in the breath whilst yet the lung is in mid-consolidation, are of most unfavourable omen. The prevalence of an epidemic such as influenza, is said to influence the termination of pneumonia in gangrene.¹¹ Fortunately, however, this termination of pneumonia in local death of tissue is extremely rare. Gangrenous pneumonia must of course be distinguished from local gangrene of lung determined by thrombosis, or embolism of a branch of the pulmonary artery. The latter cases commonly commence with haemoptysis, the consolidations in them spread from some spot neither at the apex nor base of the lung, and the

¹¹ Wilks and Moxon's *Pathological Anatomy*, 1875, p. 334.

gangrenous signs are not preceded by those severe general symptoms which attend a pneumonia likely to proceed to gangrene.

The treatment of gangrene of the lung supervening upon pneumonia must, like that of abscess, be conducted upon ordinary principles. Abundance of nourishment is required, and stimulants are in all such cases indicated.

Lastly, there are cases now and again to be met with, cases *par excellence* of pleuro-pneumonia, in which the symptoms and signs are at first those of pneumonia, but the tubular breathing soon becomes obscured and distant, vocal fremitus diminished, and vocal resonance *œgophonic* about the scapular region: in a word, the signs of moderate effusion supervene, and at the period when the temperature natural to pneumonia should fall, it assumes the hectic type with daily chills and sweats, assuring us of the purulent nature of the fluid. After a variable interval, usually of several weeks, during which the signs do not clear up, the expectoration may become odorous, or a profuse flow of foetid pus from the lungs may announce the rupture of the *pleural abscess* through the lung, or again, the empyema may point externally. No treatment is in these cases of much avail so long as the pus remains in the pleura. If it be allowed to rupture through the lung, the patient after a long exhausting illness may ultimately recover. He may also do so if the empyema be left to point externally, and this latter event would be encouraged by diligent poulticing. There can be no doubt, however, that the right treatment of any such case is the timely removal of the pus by puncture, and the employment of the exhausting syringe.

CHAPTER IV.

ON DISEASES OF THE PLEURA.

PLEURITIS, varieties of—Simple pleurisy, causes: pathology: symptoms and signs: diagnosis; treatment—Local pleurisy, sometimes simple: generally from extension—Suppurative pleurisy: compared to purulent infiltration of the lung: symptoms: physical signs: difficulties in diagnosis: views of Bacelli—Pleuritic effusion, special signs indicating excessive effusion: position of the heart: axis of heart rarely transposed: illustrative case. Pulsation of fluid—Stress of circulation in opposite lung.

THE pleura on each side of the chest is a closed serous cavity or sac intimately applied to, and in organic union by its outer surface with, the lung and the costal parietes. The internal surface of the sac is lined with smooth laminated epithelium. The costal and parietal portions of the internal surface are in close contact, lubricated merely by some moist serous secretion. This contact or apposition is maintained by atmospheric pressure bearing upon the interior of the lung and the exterior of the chest-wall, which more than counter-balances the constant tendency for the two surfaces of the sac to spring apart from the opposite elastic tractions of the lung and thoracic wall. In the cellular tissue subjacent to the pleura, and more especially to that portion applied to the lung, is a layer of lymphatics freely inosculating with the pulmonary lymphatics and communicating by open mouths (*stomata*) surrounded by glandular epithelium with the pleural sac. These lymphatics run their course towards the root of the lung.

Pleuritis—inflammation of the pleura—occurs in several forms due to several different causes. Its varieties may be conveniently classified under three headings:—

1. Simple pleurisy, } local.
{} general.
2. Pleurisy from extension.
3. Suppurative pleurisy.

I. Simple Pleurisy.—Amongst the common causes of simple pleurisy exposure to cold is the most frequent. The depression of temperature may be general, in persons insufficiently clothed, or whilst vital resistance is lessened from mental shock or insufficient food. Or the patient may receive a chill from exposure to a draught or sudden change of temperature whilst over-heated. Certain diathetic states favour the occurrence of such pleurisies, *e.g.*, the rheumatic diathesis, albuminuria. But prior to the attack the patient may have been in good health.

The *pathology* of the disease may be briefly summed up as consisting in the first place of hyperæmia of the pleura, its surface becoming minutely injected; in a very few hours the normal glistening appearance of the pleura is lost, it becomes cloudy as though breathed upon and gradually covered with a layer of lymph. Both the costal and visceral pleura are thus affected, and the effect of the movement of the two surfaces, both covered with sticky lymph, upon one another is to roughen them, causing the effused lymph to present numerous little elevations and pittings. New vessels rapidly extend into the lymph from the pleural vessels forming loops which, unless the opposed surfaces be speedily separated by effusion, meet and inosculate, and thus organic union takes place between them. But most commonly in simple pleurisy an effusion of liquor sanguinis escapes from the vessels and collects in the pleura, thus separating, more or less widely from below upwards, the pleural layers. In due time, and in normal cases, the acuteness of the disease passes, the fluid effusion is again absorbed, the lymph-covered surfaces come again in contact and rapidly adhere, the originally effused lymph becoming in course of time degenerated and absorbed.

The *symptoms* and *signs* of pleurisy are in accordance with its pathology. Pain in the side and rigors are the two symptoms which usher in an attack of pleurisy, and either may

precede the other for a few hours. The pain is that of an acute "stitch" in the side usually felt in the lower axillary or infra-mammary region, but not uncommonly referred, and especially so with children, to a much lower point in the abdominal wall to which the terminal cutaneous twigs of the affected intercostal nerves are distributed. The pain interferes with the respiratory movements, which are restrained and shallow, the patient inclining to the affected side so as to lessen its movement. The rigors are of variable severity, sometimes very sharp and decided, at other times amounting only to recurring chills. It is stated that the rigors of pleurisy are repeated, whilst in pneumonia one severe shivering occurs at the commencement of the disease: on neither hand, however, is this statement more than generally speaking correct. There is occasional dry interrupted cough. The temperature is elevated usually to about 102° or 103° , the face anxious, pale, the pulse small and moderately frequent. The fever has never the marked character of that of pneumonia, and the flushed cheek so characteristic of the latter disease is rarely present. In a word the patient with acute pleurisy is not so ill as one with pneumonia, although he may be in more suffering.

On listening to the chest the respiratory murmur will be found to be suppressed on the affected side, and more or less distinct friction sound is heard, most distinctly at the end of inspiration and the beginning of expiration, over the seat of pain, usually in the mammary or infra-mammary region or over the base of the lung. The percussion is at first unchanged, but within a short time dulness may be detected at the extreme base posteriorly, gradually extending upwards and becoming perceptible in front mounting towards the apex. This dulness varies somewhat with the position of the patient: whilst lying down, for instance, the resonance may be good to below the nipple, whereas on sitting up there is dulness at this point and, it may be, as high

as the second or third rib. Over the lower portion of the dull area the respiratory murmur is absent and the friction is no longer to be detected, but as the upper limits of dulness are approached in the scapular region distant tubular breathing may be heard, and friction sound of a moister character is audible, especially in front. The vocal vibrations are annulled and vocal resonance is generally diminished over the area of dulness, but again towards the upper limits of dulness, especially behind, the voice sound becomes intensified and altered to the peculiar nasal punch-like quality known as *cœgophony*. In association with these signs and *pari passu* with the increasing dulness, the apex of the heart will be found to be displaced more and more towards the opposite side.

In marked contrast to the effacement of respiratory sounds on the affected side, is their exaggerated intensity, on the sound side.

Where the effusion is considerable, the whole side becomes dull from apex to base, and the dulness includes the sternum extending to its opposite margin, and being continuous beyond it with the displaced cardiac dulness. At the sterno-clavicular angle of the affected side, however, a remarkable tubular quality of resonance (Skodaic resonance) is elicited on percussion, and at this point the respiration is often intensely bronchial. I have said as yet nothing of measurement as affording a sign of effusion, because only in cases of extreme effusion do we get any appreciable difference on the two sides to measurement. The eye, however, can at an earlier period detect an alteration in shape on the affected side, which appears larger inasmuch as it does not contract with expiration as the sound side does. In extreme cases the side is obviously distended, the nipple and shoulder raised, and the intercostal grooves less perceptible.

As the signs of effusion progress the pain lessens, and the patient at first feels more comfortable. The respirations be-

come quieter but they soon quicken, the patient inclines more to the diseased side and requires the head to be raised, and if the effusion increases much dyspnœa may be experienced.

The fever of acute pleurisy subsides in a few days to a week or a fortnight, according to the severity of the attack, the tongue cleans, the appetite returns, and the patient has only to gain strength and to await recovery from the results of the local inflammation and effusion. As the fluid becomes absorbed the lung expands from above downwards, the pleural friction-sound returns, and with it usually some pleuritic pain, although of not nearly so intense a character as in the first instance.

Dr. Gee, in his work on Auscultation, has very minutely and accurately described the physical signs and other phenomena attendant upon recedent effusions; suffice it here to say that the respiratory murmur slowly returns, at first in the upper portion of the chest, and the heart gradually resumes its normal position. The absorption of the last portion of fluid is often considerably delayed, and dulness and weak respiration may long persist at the posterior bases. In some cases the effusion may shew no sign of becoming absorbed, in others again complications may arise. But in the great majority of instances the *prognosis*, in cases of simple acute pleurisy is decidedly good.

The *diagnosis* of pleurisy is not attended with any great difficulty. As regards *pain* it may be simulated by pleurodynia, or intercostal myalgia, neither of which affections are, however, attended with febrile phenomena, nor with friction-sound nor the signs of effusion. Intercostal myalgia is a local affection accompanied with marked tenderness on pressure over some one or two of the intercostal spaces. In pleurodynia, on the other hand, the pain is very intense and inconstant, and occurs in a patient subject to neuralgia and who has, perhaps, had previous similar attacks. Pneumonia is the remaining disease with which pleurisy

may be confounded and we must observe that the two often co-exist. The absence of rapidly supervening bronchial respiration with bronchophony and fine crepitation, the freedom from blood-stained sputa, and the gradual effacement of respiratory sounds and of vocal fremitus, with increasing displacement of heart as dulness on percussion increases in extent, render the diagnosis of pleurisy with effusion certain. The chief difficulty in diagnosis is, however, to ascertain the probable *nature* of the pleurisy, whether simple with serous, or suppurative with purulent effusion. We will postpone the discussion of this point until our consideration of the graver malady.

The *treatment* of simple pleuritis does not usually call for any very energetic measures. The patient should be kept in bed in a warm room with the affected side wrapped in hot linseed poultices. The application of a few leeches to the side will relieve pain by subduing intensity of inflammation, and they may be very usefully employed in cases in which such small blood-letting is not contra-indicated. Further pain must be relieved and sleep produced by the aid of opium in sufficient doses, Dover's powder being the preferable form for administration. In adults, subcutaneous morphia injections may be preferred. The bowels should be well relieved, and but little further medicinal aid is needed at this stage of the disease. The temperature and the signs of effusion must be watched. If the dyspnœa become urgent, the fluid must be drawn off, otherwise the treatment of the effusion may admit of difference of opinion. (*Vide infra*, Hydrothorax.) As the fever subsides, more stimulating applications may be made to the chest, flying blisters or iodine applications to hasten reabsorption. As this absorption is being effected, and when pain arises from the pleural surfaces again coming together, the firm application of a broad band of adhesive plaster round the affected side, extending well beyond the median line in front and behind will relieve pain, and facilitate adhesion. Mineral acid tonics, usually with iron, are now

indicated, and change of air will hasten the complete reabsorption of fluid, and the restoration of the function of the lung.

Local pleurisies, although usually belonging to the next category, are sometimes simple and uncomplicated, and are then characterised by severe pain, limited to a small area over which a friction sound is audible. They are usually "dry" pleurisies, being unattended with effusion, and soon resulting in adhesion. Undoubtedly such a pleurisy may extend and become general, but it has little tendency to do so, being in the first instance unaccompanied by any severe constitutional symptoms. If the disease be detected at the apex we may very strongly suspect the presence of tubercle, and in other situations although, as I have observed, the disease may be strictly simple, it is yet much more commonly met with in drinkers, or in those who are syphilitic. A blow upon the chest, or fracture, or disease of the rib may also lead to local pleurisy.

The application of a firm band of strapping round the affected side so as to keep it at rest, will usually absolutely remove pain and speedily cure the disease. A flying blister is also often very serviceable.

The patient's general health usually requires building up with tonic remedies.

2. *Pleurisy from extension* is generally local, limited that is to say to the neighbourhood of the lesions from which it has spread. This form of pleurisy is generally dry and most commonly complicates phthisis, with which disease, indeed, it is from time to time invariably associated. Inflammatory diseases of the lung are also attended with pleurisy, pyæmic patches and haemorrhagic infarcts are always indicated on the surface by patches of inflamed pleura. Disease of neighbouring parts, aneurism, mediastinal tumours, cancer of the breast involving the thoracic wall—frequently give rise to pleurisy in their neighbourhood. These pleurisies call for

no special remarks as regards either pathology, diagnosis, or treatment. I must repeat here, however, what I have already said at an earlier part of this volume, viz., that the secondary pleurisies now referred to may arise not only by direct spread of the inflammatory disease or extension of irritation, but also, in cases of phthisis especially, by the friction of a portion of lung surface which from consolidation no longer conforms to, or follows the natural expansile movements of the chest. It is further probable that local pleuritic inflammation not unfrequently arises as the secondary consequence of congestion determined by aspiration of blood to a part which, in consequence of solidification of the lung beneath, cannot follow the expanding movements of the chest-wall.

3. *Suppurative Pleurisy*.—Suppurative inflammation of the pleura is a far graver malady than any we have yet considered in this chapter. Whereas in acute simple pleuritis the inflammatory products consist of simple fibrinous exudation which coagulates in a thin layer upon the pleural surfaces, and of liquor sanguinis which exudes through the weakened vessels and collects in the pleura; in suppurative pleurisy, on the other hand, we have a more intense and a less sthenic process, accompanied by the exudation of a more corpuscular and less coagulable lymph, and of liquor sanguinis rich in leucocytes. The one case—simple exudative pleurisy, is perfectly analogous with sthenic exudative pneumonia, the other,—suppurative pleurisy, with the corresponding disease occasionally met with under greatly depressed conditions of system whether from intemperance or other causes, viz., purulent infiltration of the lung. And just as in pneumonia the simple may pass into the suppurative inflammation, so it is not unknown in pleurisy for a hydro-thorax to become an empyema.

Modern science fails as yet in enabling us to express in more precise terms what are the conditions that determine suppurative rather than simple pleurisy, and we have to speak of “de-

pressed conditions of the system," "morbid constitutional states" and the like; expressions which, nevertheless, are in keeping with our knowledge and are generally understood. We may indeed with some plausibility maintain that some septic agent present in the blood renders the inflammation purulent rather than serous as in the joint affection of pyæmia, although the pus producing quality in the blood is very difficult to estimate. In such diseases as pyæmia, scarlatina, typhoid, and in the puerperal state, pleurisy, when it arises, is most generally suppurative. In rheumatism, gout, albuminuria, and delirium tremens, it is, on the other hand, as a rule simply serous. Empyema is again very apt to occur in cases in which disease of the lung is also present. Perforation of the pleura in phthisis almost always leads to pyo-thorax, no doubt principally from the escape of purulent matter into the sac.

The *symptoms* of suppurative pleurisy do not very strikingly differ from those of serous effusion; in many cases from symptoms and signs alone it is impossible to make a certain diagnosis. The symptoms are commonly less acute but more adynamic in character than in ordinary pleurisy. The rigors or chills are more persistently recurrent, with daily fever continuing week after week; the pulse is from the first more frequent than in serous pleurisy, and keeps up its frequency throughout the disease. The tongue is more furred with a greater tendency to become dry and brownish in the centre. The general condition of the patient is more depressed and anxious, and emaciation is much more rapid than in ordinary pleurisy. But perhaps the most important sign of the effusion being purulent, (although not absolutely characteristic), is the occurrence of hectic sweats breaking out whenever the patient falls asleep.

The *physical signs* must necessarily be for the most part the same, whether the case be one of simple or suppurative pleurisy. Are there any physical signs, however, which are characteristic of pus in the pleura? Edema of the chest wall when it occurs, is I

believe, an absolute sign of the presence of pus. Erysipela-tous blush over a portion of the chest, or still more evident pointing, are certain signs of the effusion being purulent. But these are late signs, and therefore of greatly mitigated value. It is maintained by Dr. Bacelli of Rome,¹¹ that by the mode of transmission of vocal vibrations, the diagnosis between empyema and hydrothorax may with certainty be made, whilst all other evidence of duration of disease, oedema, emaciation, intermittent fever, and anaemia are insufficient for the purpose. In order to estimate this sign, the unaided ear must be applied to some convenient point of the affected side, e.g., over the dull region posteriorly below the angle of the scapula, and the patient directed to whisper some rough word; this will be well conducted to the ear if the fluid be serous, not so if it be purulent. Dr. Bacelli's theory to account for the difference of conduction in the two cases is, that in serous effusions the fluid, being thin and homogeneous, transmits vibrations with facility, but the more the fluid departs from the homogeneous nature of serum, the thicker it is, and the more clouded by the presence of amorphous protein bodies and morphological or corpuscular elements, the less complete the conduction of sound through it. This physical sign is totally different from that of oegophony which is a compressed lung-sound, modified by transmission through a thin layer of fluid. The value of Bacelli's sign needs further confirmation than it has yet received; my own observations would lead me to regard it as an important addition to our means of differential diagnosis, but not as being of absolutely distinctive significance. There is indeed, only one criterion in these cases, and that is the exploring puncture made with a grooved needle or fine trochar, and this method may be employed without danger or inconvenience at any period at which absolute certainty of diagnosis becomes of importance.

¹¹ Archivio di Medicina, Roma 1875. See a critical reference to Dr. Bacelli's paper by Dr. Gueneau de Mussy.—*Union Médicale*. Jan. 4th, and Feb. 17th, 1876.

In cases of great effusion into the pleura, whether of serum or pus, certain signs occasionally present themselves to which we have not yet referred. The diaphragm becomes greatly depressed, the spleen and liver correspondingly lowered, and in extreme cases the depressed diaphragm may be felt (especially in left-sided cases) as a rounded tumour below the costal margin. In one case, a male patient with great effusion into the right pleura, I have observed the action of the diaphragm on the depressed side to be apparently reversed, the hypochondrium on this side sinking inwards with inspiration, whilst on the opposite side it became as usual more full. Whether this appearance was really due to contraction upwards of the depressed half of the diaphragm, or whether it was merely due to a *recession* upwards of that segment on the relief of intra-thoracic tension during inspiration I cannot say. I have somewhere read the statement of a good authority¹² that in cases of great effusion into the pleura, the tension within the pleura is increased during inspiration by the depressed half of the diaphragm (having its arch reversed on the affected side) contracting upwards instead of downwards. The question is, whether the half of the diaphragm so affected is not really paralysed; but in the case above alluded to, the patient, during the removal of the fluid suffered much from what appeared to be *spasm* of this half of the diaphragm, a fact in favour of the muscle having preserved its irritability.

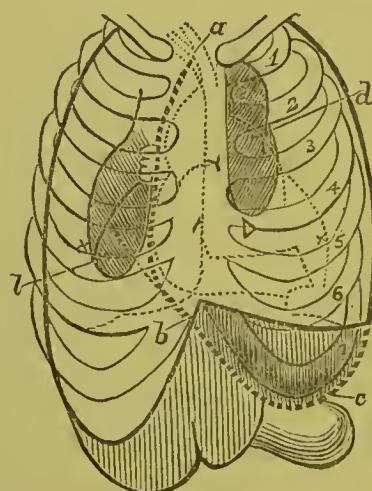
The displacement of the heart in cases of extreme effusion becomes very great, the whole organ being transposed towards the healthy side with the general displacement of the mediastinum. The axis of the heart when the organ is displaced by effusion becomes somewhat more vertical; this is best seen in left-sided cases, but only in rare and extreme cases does the axis of the heart become altered in direction beyond the vertical line. I ventured eight years ago¹³ to state this

¹² I regret that I cannot now give the reference to this author.

¹³ Notes on Displacements of the Heart.—*Brit. Med. Journ.*, July 17th, 1869.

proposition more absolutely than I do now, in opposition to the view of Dr. Williams¹⁴ and that of the late Dr. Sibson, as given in his Medical Anatomy, and maintained that neither in effusion into the left pleura, nor in cases of contraction of the right lung does the direction of the heart's axis ever become so altered. Although further experience has only confirmed me in the general truth of this statement, yet I have learned that like most absolute assertions, it does not hold good in all cases. For I have met with one instance *post-mortem* in which the heart, generally transferred to the right of the sternum, further had its axis distinctly although slightly inclined to the right. The accompanying rude sketch from an outline filled in at the autopsy represents the position of parts found on removing the sternum in the case alluded to. The case was one originally of pneumothorax, supervening ten months previously upon phthisis affecting the left lung in a girl aged 17. Purulent effusion had subsequently set in, and all signs of the original pneumothorax had disappeared. The patient died somewhat suddenly, paracentesis having been too long delayed in consequence of a natural anxiety not to

FIG. 10.



¹⁴ *Diseases of the Chest*, 4th Edit., p. 119.

reinduce the pneumothorax. At the autopsy the heart was fixed *in situ* by means of stilettes. The thorax was naturally small and long, as noted by the firm line on the diagram within that representing outline of healthy right side. The left side was much distended, the costal margin raised, the diaphragm (*b, c*) forcibly depressed, and the mediastinum (*a, b*) displaced by the fluid effusion which amounted to $5\frac{1}{4}$ pints.¹⁵ The left lung (*d*) was crowded and shrunken backwards, and, with its thickened pleural covering, weighed only $5\frac{1}{4}$ oz. "The heart's apex was opposite the upper border of the fifth rib,¹⁶ in vertical line of junction of inner and middle third of the right clavicle: base opposite lower border of second cartilage. Axis slightly to right, anterior surface formed mainly by left ventricle (*i*). The *back* and *front* of the heart directed obliquely to left and to right respectively." Thus the heart had become so twisted as to present itself obliquely, edgeways to the view of the observer in front. This disposition of the heart to turn over and to present its posterior surface forwards in cases of effusion has also been pointed out by Dr. Sibson. This is, however, the most decided case of the kind that I have met with.

In almost all cases in which the effusion is very considerable a murmur is developed over the heart, most audible over the base, but it is difficult to say in the altered position of parts what is the exact point of origin of the bruit. That the murmur is due to direct pressure, and probably to some twisting of the aorta, is obvious from the fact that, as the fluid escapes from the pleura during paracentesis, and usually after the removal of the first two or three pints, the murmur

¹⁵ This is by no means, absolutely speaking, a large quantity, but relatively to the dimensions of the thorax in this case it was excessive.

¹⁶ Represented in diagram as opposite lower border of 5th rib, but as shown by the outline the ribs were more oblique than those represented by the normal tracing. N.B. The depression of the diaphragm is exaggerated in the engraving.

disappears. The presence of this displacement murmur is an urgent indication for paracentesis.

Pulsation is sometimes communicated to the fluid by the heart's action, and in some cases this pulsation strongly simulates that of aneurism. Dr. Walshe has referred to this point, and similar cases have been observed by others, in all of which however, according to Fraentzel¹⁷, the fluid has been purulent. In each of the two cases observed by Traube, and also in the one that came under Fraentzel's notice, pericarditis with effusion into the pericardium was present. Traube considers the occurrence of pulsation as due in part to suppurative softening and hence increased extensibility of the pleura costalis, in part to the presence of pericardial effusion favouring the transmission of the heart's impulse. Some two or three years ago Prof. Maclean was kind enough to allow me to examine at Netley a soldier who had a very excessive effusion into the left pleura, and who had so marked a pulsation in the left supra-mammary region as to cause some hesitation in performing paracentesis, lest there should be a large aneurism in addition to the hydrothorax. A right judgment was, however, without much difficulty arrived at, and a large quantity of *serous* fluid was removed, and with it the doubtful sign of aneurism disappeared. There was no pericarditis suspected in this case (and the heart was of course carefully auscultated). Another case of effusion, right-sided and of older standing, came under my notice at the Brompton Hospital some months ago in which a rhythmic impulse conveyed through the fluid suggested aneurism as a complication. The fluid was, however, removed by several tappings, it was somewhat opaque and at first slightly blood-stained. No aneurism was present. It is thus clear that neither suppuration in the sac nor pericarditis is necessarily associated with pulsation, and probably nothing more is needed to account for the phenomenon in question than an amount of fluid which

¹⁷ Ziemssen's Cyc., vol. iv, p. 638.

shall exercise a certain degree of pressure (neither too much nor too little) upon the beating heart. Two or three other instances have come under my observation in which the diagnosis of mere fluid in the pleura has been much shaken by another unusual pressure effect, viz., altered quality of voice and cough. A husky voice, and a laryngeal quality of cough undistinguishable from that so often heard in cases of mediastinal tumour or aneurism, have given rise to great doubts as to the diagnosis, yet both phenomena have disappeared after paracentesis.

We may, in all cases of extreme effusion, detect signs of stress of circulation in the opposite lung, and these signs again should at once prompt our immediate interference. The respiration, naturally exaggerated, becomes blowing in quality, and accompanied by catarrhal râles, sometimes with moist crepitation from oedema. These sounds may be heard either at the base or the apex. I have mostly heard them over the upper part of the chest near the margin of the healthy lung. In one well-marked case, the sputa, frothy and adhesive, became streaked with blood. All signs, however, disappeared on removal of a large purulent effusion.

The questions respecting the operative treatment of hydro- and pyo-thorax may best be considered in a separate section; they have given rise to much controversy, and many practitioners must feel somewhat hampered and embarrassed in action by so many, and often conflicting opinions.

CHAPTER V.

ON THE TREATMENT OF PLEURITIC EFFUSIONS.

SIMPLE inflammatory effusions: indications for paracentesis: methods of operating. Chronic Hydrothorax—Empyema: necessity of paracentesis: methods of operating, (1) antiseptic, (2) by single opening and injection of pleura, case related, (3) double opening and drainage—Chronic empyema, treatment of, case related.

THE relative value of the various methods of treatment of different kinds of pleuritic effusions, has been so frequently and so fully discussed both in this country and abroad, that one would think the time had come when something like an uniformity of opinion and practice should prevail. It is far from being so, however: only quite recently nearly opposite views as to the value of thoracentesis in serous effusions have been advocated by equally distinguished observers.

In the following remarks I cannot attempt to deal with the subject critically, and a mere review of the opinions of others would be beside the practical scope of the present work. I will therefore, content myself with relating those methods which have proved most successful and trustworthy in my own experience.

Pleuritic effusions are naturally grouped into.—

(a.) Simple inflammatory effusions, in which the fluid is sero-fibrinous, and which may be acute or chronic.

(b.) Suppurative effusions, in which the fluid is purulent, also of recent or old date.

(c.) Hydrothorax or dropsy of the pleura.

(a.) *Simple inflammatory effusion into the pleura.*—It is within the experience of most practitioners, that in the large majority of cases of acute effusion into the pleura the serous fluid is again absorbed with great rapidity. In such cases then no operation is needed in the acute stage, and, more than this, to meddle too hastily with such cases is highly injudicious, for the whole surface of the pleura, both parietal and

visceral, is inflamed, and the presence of fluid is the surest and most natural means of giving that rest to the inflamed parts which they need. The effusion reaches its acme, the fever subsides, and in a few days the tide turns, reabsorption being effected in a few weeks.

All these cases must, however, be keenly watched, "Nature" must not be blindly trusted always to effect what, if needed, can be so readily accomplished by Art. One might indeed, feel some misgivings as to whether Nature ought ever to be burdened to remove so vast an accumulation, did not experience show that she is fully equal to the task. Nor should there be any real difficulty in understanding this ready removal of fluid collections, when we consider that throughout the body, secretion and absorption are perpetually going on, constituting an extra-vascular circulation amounting in the aggregate to many pints. How large a quantity of fluid is daily secreted into the digestive tract to be again absorbed is well known. In the cellular tissue also, and in the serous cavities, a simpler form of the same process goes on *guttatim*, absorption keeping place with, and counter-balancing effusion. But in dropsy of either of these spaces, exhalation rapidly proceeds, whilst absorption is in abeyance, and by and by these processes may be reversed with no great strain upon the system.

If, however, at the lapse of a week or ten days from the time when the pleuritic effusion has reached its height, appropriate remedies having been used, there be no signs of any material abatement of the fluid, it will be judicious to draw off a portion of it. Doubtless the employment of purgatives, and diuretics or mercurials would still in most cases ultimately effect the withdrawal of the fluid through the natural channels. I have known an acute serous effusion require six months treatment by tonics and diuretic remedies for its final removal. In another case after many months of fruitless treatment by ordinary methods, mercury, pushed to salivation, rapidly removed

the effusion.¹⁸ But this protracted or severe medicinal treatment is not always successful, and is at least attended with great risk of permanent damage to the lung, and of complication with other diseases. The better course in such cases surely is to adopt the timely removal of a portion at least of the fluid by paracentesis; but the worst course of all is to leave such cases alone or to treat them with indifferent drugs. Dr. Bowditch observes "I cannot see any valid reason for continuing any active treatment more than one, two, or three weeks without puncturing."¹⁹

In those cases in which there is an indisposition on the part of the vessels and lymphatics to reabsorb the fluid, the effusion is commonly very great, as shown by the prominent side, the raised shoulder, the displaced heart, and, frequently, the presence of a cardiac murmur. The intra-thoracic pressure is sometimes very considerable. I have ascertained it to be as high as an inch or an inch and a half of mercury.²⁰

Now although during the inflammatory fever fluid can, even up to this degree of pressure, be effused, owing to the high tension of the general circulation, and the relaxed condition of the vessels locally, yet when the fever has passed, a more or less general exhaustion and anaemia prevails, the pressure of the circulation falls, and the effect of the high pressure upon the pleural surface is necessarily to render it especially anaemic, and, by the powerful compression of the lung, to hinder absorption by the lymphatics. The fact, frequently noticed, that after removal of even a small portion of the fluid the rest is rapidly absorbed, almost demonstrates the correctness of this view. Trousseau observes "this greater slowness in the absorption is, perhaps, as much dependent on

¹⁸ Dr. Hope—*Medico-Chirurgical Review*, 1841, strongly recommended the treatment of pleuritic effusions by mercury.

¹⁹ *Thoracentesis* by Henry J. Bowditch, M.D., 1870.

²⁰ "Observations on Paracentesis," *Clin. Soc. Reports*, Vol. iii, 1870.

Med. Chir. Trans., Vol. lix, p. 187.

the pressure exerted by the excessive quantity of fluid upon the serous membrane by which absorption has to be performed, as by the mere greatness of the quantity."²¹ It is probable that absorption of pleuritic effusions takes place largely through the lymphatics, one would otherwise expect to find some albumen in the urine, whilst so large a quantity of albuminous fluid was being taken up by the systemic veins. This I have not found, and Dr. Dickinson remarks in his recent work on albuminuria, "It has been said that the same result (temporary albuminuria) has followed the rapid absorption of serous fluid from the pleura, a sequence, which, to say the least, must be rare."

Chronic pleuritic effusion.—Patients may live for a long time, and with surprisingly little discomfort, with very large effusion into the pleura. Not a few cases are still from time to time met with in which one side of the chest is distended with fluid, the effusion dating from an attack of pleurisy months or even a year or two previously, and which, on removal, proves to be perfectly limpid and serous. I have myself seen such cases of eighteen months and two years duration, and think with Dr. Wilson Fox,²² that the fear lest the effusion should become purulent from mere lapse of time, urged by Troussseau as a reason for early operation, is not well grounded; there is very little disposition for such a transformation to take place, unless the patient have a fresh inflammatory attack. But perfect recovery in these old standing cases is well nigh impossible, the lung having become bound down and thickened by long continued compression, and it is to be hoped that the reproach of their not very infrequent occurrence will soon be removed from us.

In some other cases, absorption goes on well enough up to a certain point, beyond which, however, the patient remains at a standstill, his chest perhaps half full of fluid. In these cases there is usually considerable depression of general health, and iron tonics, good diet, and iodine embrocations or blistering,

²¹ *Clinical Medicine.* Syd. Soc. Transl. of 3rd Edit., 1868, p. 229.

²² *Brit. Med. Journal*, December 1877, p. 752.

with change of air, will generally effect complete absorption. If sure that the fluid be not purulent, one would be loth to interfere at this stage.

Finally, at any period of effusion into the chest, danger may threaten the patient, (*a*) from cardiac syncope, (*b*) from asphyxia, (*c*) from exhaustion, and in all such cases prompt interference is needed.

By way of summary it may then be said that the points to be taken into account in deciding upon the necessity of performing paracentesis in cases of serous effusion are as follows:—

1. *At the end of the first week.*—The effusion will probably not yet have reached its height, and the fever will not have subsided, the acuteness of the inflammation not yet having abated. It would, therefore, *save in the presence of very urgent symptoms*, be injudicious to interfere by any operative measure at this stage.

If, however, suffocative or syncopal symptoms threaten, a small quantity, a pint or two pints, of fluid must be withdrawn. More than this should not be attempted for fear of inducing haemorrhage into the pleura, or of setting up afresh active congestion or inflammation, with the additional danger of the new attack being of the suppurative kind.

2. *At the end of the second week.*—The presence of urgent dyspnoea, cardiac bruit, congestive râle on the healthy side, paroxysmal cough, decided lividity, or other signs of excessive and dangerous pressure, would render the operation not only advisable but imperatively necessary without a moment's delay. In the absence of any urgent symptoms, however, such diuretics as acetate of potash, iodide of potassium, digitalis, infusion of broom, &c. would be used, an occasional brisk purgative administered, and iodine frictions or flying blisters applied to the chest.

3. *At the end of the third week.*—The evidence that a large effusion is not abating under ordinary absorbent treatment

would be sufficient to render advisable the removal of some of the fluid by paracentesis. In deference to the opinion of some physicians, another week might be allowed, but my own opinion would decidedly favour the earlier operation.

PARACENTESIS THORACIS IN SEROUS EFFUSIONS.

The first thing to be done, having decided upon paracentesis, is to select and mark the spot for puncture. In uncomplicated cases *the sixth or seventh space about the mid axillary line* is the best. But in all cases, even in those apparently the most simple, the position of the liver, heart and diaphragm must be precisely ascertained, and the absence of vocal fremitus and of respiration at the spot chosen satisfactorily made out.

Dr. Garland, in his recent work on pneumono-dynamics,²³ demonstrates by a series of most interesting experiments that "the diaphragm does not bag down in any case, until the entire weight of the injection exceeds the supporting force of the lung." That is to say, until the elasticity of the lung which maintains the diaphragm in its arched condition, is completely neutralised by the effusion. Traube²⁴ also refers to a crescent-shaped region of tympanitic resonance at the anterior base in left-sided effusions, which disappears as the effusion, becoming greater, forces the diaphragm downwards. The practical outcome from these experiments is, that we may, even with considerable effusion, have the diaphragm still arched upwards: on the left side to be recognised by a tympanitic quality of resonance, on the right side by the position of the liver. The heart, however, will be already displaced, again showing that this displacement is not necessarily significant of intra-thoracic pressure. Bulging downwards of the diaphragm is, however, evidence of pressure.

²³ *Pneumo-Dynamics* by G. M. Garland, M.D., New York, 1878, page 51.

²⁴ *Gesammelte Beiträge* ii. p. 857, referred to by Fraentzel, Ziemssen's *Cyclopædia*, Eng. Trans., who speaks of the resonant space mentioned as "the half-moon shaped region."

A vertical and horizontal mark should now be made upon the chest indicating the precise spot chosen.

The patient reclining near the edge of the bed, with the head and shoulders slightly raised and leaning towards the diseased side, a piece of ice with a flat surface an inch or two square dipped in salt should be applied with firm pressure to the spot marked for puncture. In twenty or thirty seconds the spot will be frozen; it should then be rapidly wiped, a small incision made through the skin, and the trochar, previously dipped in carbolised oil, smartly thrust into the pleura. The canula will have been previously connected with three or four feet of tubing filled with water and leading to a vessel containing a certain quantity of water which must be lowered two or three feet below the level of the patient; or the tubing may be connected with a bottle which can be *gradually* exhausted of air by a suitable syringe. The operation having been completed, the canula must be withdrawn rapidly, the edges of the wound being compressed between the finger and thumb and a fold of lint soaked in collodion applied. A band of strapping three or four inches broad and of sufficient length, should then be firmly applied round the affected side, so as to extend a couple of inches beyond the median line in front and behind.

The following list includes all the apparatus needed for the performance of paracentesis in a case of serous effusion, and suggests one or two further remarks respecting the performance of the operation.

1. A lump of ice and some salt.
2. A small scalpel or lancet.
3. Carbolised oil (linseed oil containing $\frac{1}{40}$ carbolic acid).
4. Trochar and canula connected with three feet of tubing and the whole previously filled with water.
5. Basin containing one pint of water.
6. Lint. Collodion.
7. Piece of strapping plaster four inches by twenty.

1. The plan named in the text of cutting a plain surface upon a lump of ice, dipping it into some salt, and then applying it firmly to the surface of the skin, is by far the best means of obtaining local anaesthesia for paracentesis thoracis. If no ice be obtainable, the ether spray may be used, or the operation performed without the aid of any anaesthetic.

2. It is always best to make a small incision through the skin (except perhaps when quite capillary instruments are used). The clean cut wound heals much more readily than the bayonet thrust, and but little pain is felt by the patient because the skin is not stretched by the trochar.

4. The best instrument for tapping the chest is the simplest one possible which will effect the object required, *viz.*, to remove fluid without admitting air from a cavity with walls more or less elastic, and which is contracting and expanding alternately, the contraction and expansion-movements increasing in depth and force as the fluid is withdrawn.

If a simple hydrocele trochar be used, it is inevitable that before any considerable quantity of fluid has been withdrawn, air will be sucked into the chest. A glance at the lower tracing at page 258, giving the intra-thoracic pressure during paracentesis, will enable us to see that a little extra depth of inspiration, as during cough, (A, Fig. 14) or even tranquil breathing (B.C.), may suffice to draw air into the chest whilst there is still positive fluid pressure present.

With regard to the question as to the expediency, I should rather say the imperative importance, of avoiding the admission of air during paracentesis in the cases now under consideration, one fails to see the usefulness of any further discussion. True it is that, in some cases, air has been admitted into the pleura without any harmful result, but these cases are exceptional, and against them must be brought other cases far more numerous, in which renewed fever, fresh pleurisy, and the conversion of the serous into purulent effusion has followed. And, apart from these directly evil conse-

quences, let us remember that air is more foreign to the pleural cavity than serum, that whilst air is present it is quite as impossible for the lung to expand, or for the heart to return to its normal position as though fluid were there, nor is the absorption of air by the pleura when altered by recent or old inflammation more easy than that of serous fluid. Why then allow this, at best, foreign, and in most cases noxious element to enter at all? The only plausible answer is, that unless we do so, all the fluid cannot be removed. This is true as a statement, but it is no answer, because we do not wish, in any case of simple effusion into the pleura, to withdraw all, or nearly all, the fluid, but only a sufficient quantity to relieve pressure and to encourage the absorption of the remainder.

Hence, then, we must have some addition to the ordinary canula by which, whilst the fluid is allowed to flow from the chest, air is absolutely excluded from entering it.

The simplest contrivance for this purpose is perhaps that ingeniously suggested by M. Reybard, *viz.*, to tie on to the neck of the canula a piece of gold beater's skin rolled into a tube. When wet, this acts as a perfect valve, allowing of the free exit of fluid but collapsing over the orifice of the canula on the slightest aspiration towards the pleura. An ordinary hydrocele trochar can be readily fitted with this form of valve. But a piece of india-rubber tubing attached to the canula, and having its other extremity under water at a lower level than the chest, is the most complete means of excluding air, and we can in this way, moreover, employ whatever degree of siphon power we wish to aid the removal of the fluid.

The trochar now generally used at the Brompton Hospital for this purpose, is one which I brought before the notice of the Clinical Society in 1870,²⁵ in the paper already referred to, and I have not since found anything to improve in its construction.

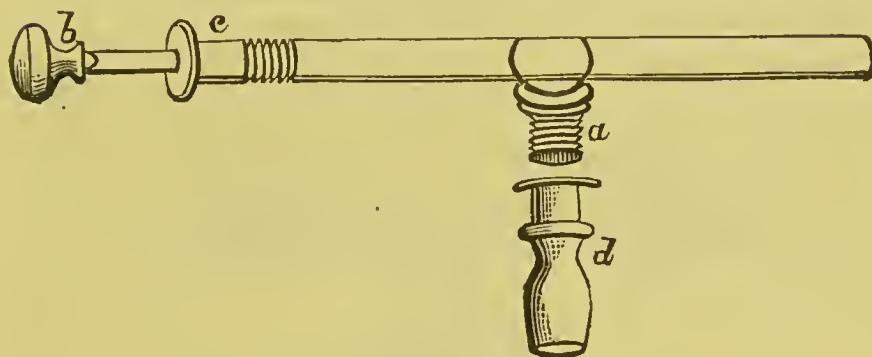
This trochar is, as I was not then aware, essentially the

²⁵ *Loc. cit.*

same as that designed by Mr. Charles Thompson, and described in the *Medical Times* in March 1858. The padding in the cap of the canula through which the stem of the trochar works seems an improvement, it was suggested by Mr. Hawksley the maker of the instrument.

The canula (Fig. 11) has a calibre of about $\frac{3}{16}$ of an inch

FIG. 11.



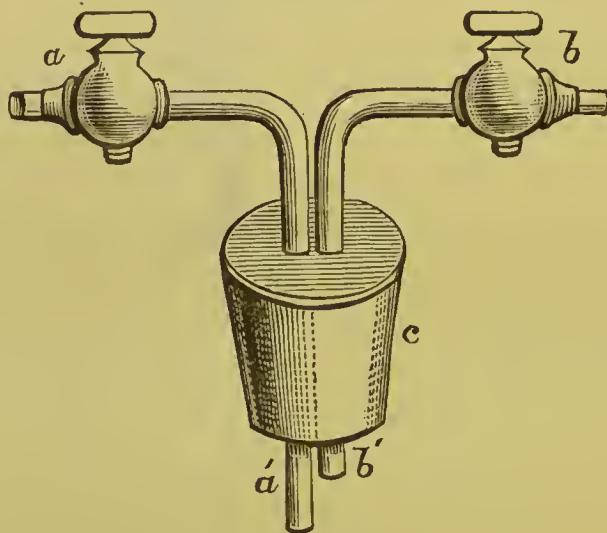
(4 mm), and is provided with a lateral branch (*a*) furnished with a leather collar on to which the tube piece (*d*) screws. The trochar has only a small and light handle, and works through the cap of the canula: this cap (*c*) is so padded with leather that, when forcibly screwed down, the stem of the trochar is grasped by the padding in an air-tight manner. The tubing attached to *d* should be about three feet long, and should be interrupted, near the canula, by a piece of glass tubing let in so that the nature of the fluid &c., may be observed. It is a very good plan to fill the tubing, ready attached to the canula, with water before making the puncture. As soon as the instrument has been introduced, and the stilette (*b*) withdrawn, the fluid flows through *a* and no admission of air is possible. The trochar should be dipped in carbolized oil before being used, and the receiving basin lowered so as to allow a siphon column of between two and three feet of tubing.

It is often convenient, and if properly managed quite harm-

less to withdraw the fluid into a bottle by aspiration. But I believe, that in all cases of paracentesis what is commonly understood by aspiration is unnecessary and dangerous.

For the purpose now indicated, the fittings of Potain's aspirator are the best. A cork (*c*, Fig. 12) made of india-rubber, and of such dimensions as to accurately fit any ordinary wine or seltzer-water bottle, is perforated by two tubes, one of which (*a*) fits on the basin end of the exit tube attached to the canula already described. The second tube *b*, is adapted to an

FIG. 12.



exhausting syringe. Each of these tubes is supplied with a stopcock, and the tube *a* in connection with the canula should extend somewhat further into the bottle than *b'*.

Now the ordinary "aspiration" method is, having fitted the apparatus, to exhaust the air from the bottle, and then having connected with the canula-tubing, to open stopcock *a* when the fluid rapidly fills the bottle. The stopcock *a* is then again closed, the bottle detached and emptied, and the process repeated.

By adopting this method of practice, however, we are employing quite an unknown suction power, which may amount to anything short of 15 lbs. to the square inch, and which is

not only quite unnecessary but, if incautiously used, extremely dangerous, and especially so towards the latter part of the operation when there is already negative pressure within the pleura.

A very safe and useful way of using this apparatus is that which I sometimes employ, viz., to open the stopcock *a*, and just to keep the syringe in sufficient action to permit or encourage the flow of fluid through the exit tube *a* into the bottle. When the bottle is filled, *the stopcock (a) must be closed* before the cork is withdrawn, otherwise, there will be a rush of air into the chest. I cannot say that this plan has any advantage over the syphon, both methods are very convenient and safe for removing fluid from the chest, the syphon excelling the other in the uniformity and measurability of the power employed, and also in simplicity and freedom from extra fittings.²⁶ If a soft plug of fibrine block the canula it may be impelled through the tube by a momentarily increased aspiration power, or it may be dislodged by reversing the syphon or sharply thrusting back the trochar.

In cases in which fine capillary trochars are used aspiration is necessary, and the usual method of employing this agency does not remove the fluid with the same dangerous rapidity as through larger instruments, but there is the same uncertainty as to the amount of negative pressure induced within the thorax. In some cases, however, in which there is doubt respecting the diagnosis, a fine exploratory trochar would be first employed.²⁷

²⁶ It would be very easy of course by a pressure guage fitted into the bottle to know at any moment what degree of aspiration is being used, but this arrangement would require a special bottle, whereas a great advantage of Potain's cork adjustment is that it renders any ordinary bottle available.

²⁷ It would seem needless to add a word of warning on the importance of seeing to the scrupulous cleanliness of trochars or aspiration needles used in paracentesis. One case has, however, come under my own observation in which carelessness in this respect apparently led to decomposition of the fluid, suppurative pleurisy, and ultimately to the death of the patient.

7. The usual plan of applying a bandage round the chest after paracentesis is not a good one, it hampers the movements of the healthy side and distresses the patient. It may be a question whether any application of the kind is necessary, but I have always preferred to keep the affected side at rest by the firm application of a band of strapping round the lower ribs to beyond the median line in front and behind.

In acute pleuritic effusions the operation of paracentesis has rarely to be repeated, the rest of the fluid being absorbed without much difficulty.

In cases, however, of what may be called *chronic hydrothorax* (cases which will become more and more rare as acute pleuritic effusion is more efficiently treated), the lung is bound down by adhesions more or less strong. The inflammatory attack leading to such an effusion is sometimes only marked by slight symptoms, the rapid effusion no doubt relieving the inflammatory condition and certainly removing the pleuritic pains. Trousseau remarks that these patients are frequently quite unconscious of having anything seriously the matter, although the pleura on one side may contain an enormous quantity of fluid. The fluid may, as already stated, remain quite clear and limpid for many months. In other cases it is turbid and greenish, containing a certain quantity of granular "pus" cells, which are probably, however, merely effete exudation cells or leucocytes.

In chronic hydrothorax repeated tappings are necessary, and they should be performed in the way above described and with the same scrupulous care to avoid admitting air. In most cases the lung partially expands, and is partly met by some flattening of the chest wall, attraction of the heart, and enlargement of the opposite lung. Meanwhile no symptoms arise from the operation, and after a rest of a day or two it is better to allow the patient to be about and to enjoy the fresh air. Tonics and iodine frictions may be employed in the intervals, and a pad of spongio-piline may be inserted underneath the strap-

ing in the infra-mammary region, so as to keep up some pressure at this portion of the chest where flattening first commences.

EMPYEMA.

Cases of suppurative pleurisy resulting in pyo-thorax are necessarily of far graver character than those we have just been considering, and present many more points of difficulty in their treatment.

There is good reason to believe that in some rare instances, especially in children, the purulent fluid has been absorbed, the pus cells having first undergone fatty degeneration.²⁸ In some other cases the fluid portion of the effusion is absorbed and the remainder thus inspissated is deposited in a thick layer on the pleura. The spontaneous disappearance of such effusions is, however, too uncommon to be expected, and the process of reabsorption is one too full of peril to be anticipated with anything but dread. It is indeed an attempt at such absorption that occasions the most characteristic hectic symptoms in the second stage of suppurative empyema. In the event of the more liquid portions becoming absorbed and inspissated pus left behind, although for a time the patient may do well, yet at any future period pleuritic abscess or secondary tuberculosis may ensue upon the softening down of the caseous deposit.

If left to run its natural course, a case of purulent effusion will in the infinite majority of instances either (1) prove fatal from exhaustion or syncope; or (2) after a few weeks of hectic the empyema may suddenly burst through the lung and the patient expectorate a large quantity of pus; or (3) the pus may

²⁸ Dr. Wilson Fox, *Brit. Med. Jour.*, Dec. 1877, Dr. Clifford Allbutt *idem* p. 797, and Dr. Goodhart p. 727. Dr. Fox regards M. Moutard Martin (*De la Pleurésie purulente*) as having placed this question beyond dispute. I have myself seen one case which has satisfied my mind on the point as to the possibility of local empyema becoming absorbed, but I know not the ultimate fate of the patient, a child aged about 5 years.

point and discharge by a fistulous opening through an intercostal space. It is contrary to all the principles of enlightened treatment passively to allow either of these phenomena to occur, and we can never do so without taking great blame to ourselves afterwards when it is too late. In no other situation in the body would a large collection of pus be allowed to remain after it had been fairly recognized, and we should not depart from one of the best established of all rules in Medicine because statistics bring out the fact that the results of paracentesis in empyema are at present deplorably bad. We should rather scrutinize with increased diligence our methods of operating, and adopt with still greater care all those precautions of which experience and research have proved the value. For notwithstanding the fact that some cases have been recorded of recovery from empyema without any surgical help, yet the prognosis in such cases is practically almost hopeless.

If we turn to Dr. Goodhart's tabular analysis of 77 cases of empyema treated at Guy's Hospital and at the Evelina Hospital,²⁹ we find that of these, 15 were subjected to no surgical treatment and did not spontaneously discharge, of which 2 (both children aged 5 and 2½ years respectively) recovered, 2 (both adults) remained invalids and 11 died, some, it is true, with other complications besides the pus in the pleura.

In eleven other cases out of the 77 the empyema pointed and either discharged spontaneously or was incised. This kind of evacuation is equivalent to that by an imperfect and too long deferred surgical operation. In only two of these cases can a good result be said to have followed, as will be seen on glancing at the subjoined extract from Dr. Goodhart's table:—

i. (No. i). F., æt. 5 years; left-sided empyema, 1 month duration; spontaneous opening discharging 3 months, pus previously expectorated. Result; good vesicular breathing after 5 months illness.

²⁹ Empyema and its treatment, *Guy's Hospital Reports*, 1877, p. 183.

2. (No. 2). M., æt. 21 years; right-sided empyema, 2 years duration, spontaneous opening, discharging 2 years. Result; discharge continuing with retracted side, large liver and probable phthisis (some haemoptysis at the commencement of case).

3. (No. 3). M., æt. 6 years; left-sided empyema, 3 months duration, spontaneous pointing, incised, discharged for 6 weeks. Result; side much retracted, "cure."

4. (No. 25). F., æt. 6 years; left-sided empyema, 6 weeks duration, spontaneous pointing, incised, discharged one month. Result; much shrinking of chest, feeble vesicular breathing after one month. (Good).

5. (No. 30). F., æt. $4\frac{1}{2}$; left-sided empyema, 2 years duration, spontaneous pointing, incised, discharged for 6 months. Result; still discharging.

6. (No. 35). M., æt. 32; left-sided empyema, 14 months duration. Fistulous opening of 10 months duration. Result; Fistula still discharging freely.

7. (No. 37). M., æt. 18; left-sided empyema, 12 months duration, sinus discharging on admission, continued 12 months. Result; left chest retracted, heart displaced, bronchial breathing.

8. No. (38). M., æt. 25; left-sided empyema, 12 months duration, sinus discharging on admission, continued 9 months. Result; chest contracted, no vesicular breath-sound, nails much clubbed.

9. (No. 39). M., æt. 10; right-sided empyema, 2 years duration, sinus discharging on admission, continued 18 months. Result; respiration bronchial and deficient, clubbed fingers, much albuminuria.

10. (No. 40). M., æt. 20; left-sided empyema, 13 months duration, sinus on admission, discharging 12 months. Result; much flattening, amyloid viscera.

11. (No. 41). F., æt. 21; right-sided empyema, spontaneous opening, incision, counter incision and drainage, discharging

7 months. Result; chest flattened, much cough, wounds still discharging.

Dr. Barlow and Mr. Parker in their "Notes on Pleuritic effusion in Childhood,"³⁰ observe "with respect to spontaneous evacuation by external opening, our experience does not supply us with a single really good result."

Whatever then may be our ultimate method of treating cases of empyema it is perfectly clear that we must adopt some surgical measures, or take upon ourselves responsibility for a large mortality.

It is still more true with suppurative than with serous effusions, that the greater the duration of the effusion the more difficult is its ultimate removal, because in the former cases we must evacuate all the pus from the pleura, and the more complete the power retained by the surrounding walls of the cavity left behind of closing in and obliterating it the better. Hence, in active cases, as soon as it is ascertained that there is pus in the pleural cavity, the question of its removal should be at once considered.

I have already referred to the signs upon which the diagnosis of suppurative as distinguished from serous pleurisy may be made, (see page 219). The association of the attack with pyæmia, scarlatina, the puerperal state, typhoid fever, the scrofulous diathesis, or already existing pulmonary disease, renders it *much more probable* that the fluid effused will be purulent. A typhoid character of symptoms, with great rapidity of pulse; daily elevations of temperature, continued beyond the period at which in simple pleurisy it should subside; continued rapidity of pulse; the occurrence of hectic sweats and of *secondary chills*; marked emaciation, anorexia and furred tongue are all signs which strongly point to pus in the pleura. œdema of the side sometimes appears as a later sign, which when it occurs is of great importance.

³⁰ Read in the section of Medicine at the Annual Meeting, British Medical Association, August, 1877.

It is impossible to lay down any hard and fast rule, as to the exact time at which all such cases should be interfered with. No two cases are precisely alike, and each must therefore be dealt with on its own merits and in the discretion of the observer. But the general rule laid down by Dr. Hamilton Roe³¹ who (with Hughes and Cock of Guy's Hospital and Troussseau in Paris) practically revived the operation of paracentesis thoracis, should be acted upon, viz., "that as soon as it is clear that pleurisy is subdued, and that a large quantity of fluid remains in the chest, we should proceed at once to ascertain its quality by introducing the exploring needle³² (invented by Sir B. Brodie), and if it is found to be purulent, the operation should forthwith be performed." The period for exploration here referred to by Dr. Roe would be from about the second to the third week of the disease.

The next question is as to the best method of removing the fluid. It may sometimes be convenient, and particularly so in cases in which asphyxial or syncopal symptoms are threatening, to remove a few pints of the pus by means of the siphon or aspirator. But, although by this means temporary relief is obtained from any pressure-symptoms that may be urgent, the effusion will certainly re-accumulate, and we cannot be said to have yet dealt with the case until the pus has been thoroughly evacuated. Dr. Clifford Allbutt's objections to incomplete tappings or partial aspirations in purulent effusions, seem to me to be very valid ones. He remarks³³ "my two objections, and these complete ones to partial aspiration are:—1. Aspiration does not prevent the formation of a pulmonary fistula:—2. It does not prevent absorption but rather favours it. By the presence of a full cavity absorption

³¹ *Medico-Chirurgical Transactions*, 1844, p. 222.

³² The ordinary subcutaneous injection syringe (I think first recommended for this purpose by Dr. Ringer) or the needle of an aspirator would now be substituted for the old-fashioned groove needle.

³³ *British Medical Journal*, Nov. 1877, page 727.

is often prevented and fever absent; draw off some of the pus, you relieve pressure and absorption begins." These objections hold good in acute purulent effusions. In chronic empyema of old standing the conditions present are different.

Dr. Allbutt also observes, "if I have one conviction in Medicine more urgent than another it is this; if pus or septic material be present in the body we must not rest until it is removed. I therefore dislike and reprobate all temporising with an empyema." The decided views that have also been expressed by Dr. Bowditch and the late Dr. Anstie, show that the opinions of Rousseau and Hamilton Roe are weightily approved by modern authors. The very recent and valuable observations of Dr. Wilson Fox on the subject, whilst certainly giving a note of warning against hasty or rash procedure, to my mind only serve to emphasize the importance of increased attention to the minutest details of an operation, which has too often been performed carelessly.

The object of the proposed operation is quite different from that of paracentesis in simple pleuritic effusions. Whereas in the one case (simple effusion) we only desire to withdraw a sufficient quantity of fluid to relieve pressure, and to hasten the absorption of the remainder, in purulent fluid on the other hand, our object is to completely evacuate the pus, and yet to allow as little subsequent suppuration in the sac as possible.

It is clear that we cannot empty the pleura even in recent cases, without admitting air in place of the fluid. Hence we must adopt one of three measures.
1. Either to disinfect the air admitted into the pleura and, having inserted a drainage tube, to close the wound with antiseptic dressings, to be renewed with the same precautions every three or four days.
2. To make a double opening and introduce a drainage tube, so as to permit of the escape of pus as rapidly as it is formed.
3. To make a single free opening and insert a tube through which the pleura can be daily washed out with some disinfecting fluid.

Antiseptic operation.—Of the three methods the first is to be preferred and it is on the whole the least troublesome, since, although there are several points of detail needing attention during the operation and at the subsequent dressings, these dressings require to be removed at less frequent intervals, and all injections of the pleura are dispensed with.

I will briefly give the headings of this method, which the practitioner will have no difficulty in supplementing as soon as the principal objects in view are clearly discerned.

(a.) The apparatus required consists of—1. A Lister's hand-ball spray producer charged with carbolic acid solution 1-40, and having a piece of linen wrapped round the end of the tube in the bottle, so as to filter the solution and prevent the tube from becoming blocked with any foreign particles.

2. A lump of ice and some salt, or the ether spray, to freeze the spot chosen for the incisions.

Note.—It must be one person's duty to produce local anaesthesia³⁴ and then to keep a fine spray of carbolic acid constantly playing upon the part from a distance of about two feet, so as thoroughly to disinfect the atmosphere about the incisions. The spray must not for a moment be intermittent whilst the part is exposed, and the surgeon should have at hand a "guard" i.e. a double fold of linen steeped in some carbolic acid lotion, which can be applied over the wound to protect it at any time if it be necessary to interrupt the spray.

3. A saucer containing carbolized oil, 1 carbolic acid to 10 olive oil, with which trochars &c. can be anointed before being used.

Note.—The most scrupulous care must be taken beforehand to ensure that the trochars or other instruments likely to

³⁴ Although not necessary it is often highly convenient, especially in nervous people, to administer chloroform. And in those cases in which neither dyspnoea nor lividity are marked, nor the heart's action greatly weakened, the drug may be administered with as much safety as for other operations.

be wanted are thoroughly clean. All such instruments must be well washed in a strong solution (1-20) of carbolic acid before being used. Any drainage tubing should be soaked in this solution for some hours before hand.

4. A bistoury or large-sized trochar and some gum elastic catheter or wire drainage tubing securely threaded with silk.

5. Antiseptic gauze, not less than eight layers thick, and about 12 by 16 inches area. Under the outermost layer of the gauze, a piece of thin prepared mackintosh must be inserted, taking care that it does not reach beyond the edges of the gauze in any direction. Some rollers for bandaging made of the same antiseptic gauze material must be ready to keep the application in place.⁸⁵

(b.) The spot for puncture will have already been determined upon—probably the seventh or eighth space in the posterior axillary line, and the operator now (the surface having been first washed with carbolic solution 1-40 and then frozen) makes an incision through the integuments, and the spray being in full play, thrusts his bistoury or large trochar, previously rendered aseptic, into the pleura, and makes a sufficient opening to ensure the free exit of the pus.

Note.—If there be any doubt as to the eligibility of the spot chosen for puncture, a fine exploring trochar can easily be first inserted. I am inclined to think, for one or two reasons, that a large trochar is better than a bistoury for evacuating the pus in empyema, for, if smartly introduced, it is thrust clean through any false membranes, and at once secures a clear free passage for the fluid. A bistoury, on the other hand, is more apt to carry false membrane before it, or to make an incision in the membrane which, on the withdrawal of the instrument, fails exactly to correspond with that through the thoracic wall: thus the wound is practically closed by a valve, and the exit

⁸⁵ The materials are sold by Gardiner of Edinburgh, Mayer and Meltzer of Great Portland Street, and Krohne and Seseman of Duke Street, London.

of pus prevented. I have seen a striking instance of this occur to an experienced operator. The drainage tube can be readily introduced through a canula which is then withdrawn over it. Of course, however, cases differ much, and the surgeon will be guided by the amount of intercostal space at his disposal and other circumstances in choosing any particular instrument.

During the rapid escape of the fluid, the patient may become faint, in which case the flow must be temporarily stopped and brandy given.

All the fluid having been removed, a piece of drainage tubing some two or three inches in length is inserted and thoroughly secured by the ligatures threaded on to it. The tube is introduced so that its head is almost flush with the surface of the chest, the ligatures being attached to separate pieces of plaster affixed in the neighbourhood of the wound.

A small bung or cork may be now inserted into the tube, a double fold of carbolized lint applied over the wound, the antiseptic gauze super-imposed and kept in place by the carbolized bandages, special care being taken to see that the margins of the gauze are well in apposition with the chest wall. Some arrangement of shoulder straps stitched to the bandages must be devised to prevent them from slipping down.

Note.—It is advisable before applying the gauze dressing to wet the surface intended to be applied to the skin, by allowing the spray to play upon it for a few seconds.

The air is thus completely excluded, except that first admitted under the spray and what small quantity may subsequently filter through the antiseptic dressings.

The patient must be kept in bed, and the strength well supported by nutritious and digestible food. The temperature should be carefully watched, and will be found, if all has gone well, to fall to the normal. On the third day the dressings should be removed under the protection of the carbolised

spray, the plug removed from the tube, and if necessary, the tube itself withdrawn so as to ensure the escape of any collection that may have formed. A few ounces of pus will escape which should be entirely free from odour. A clean tube ready prepared must be introduced, fresh carbolised dressings applied with the same care as before, and the patient again left for three or four days according to the amount of discharge, *the object being to keep the pleural sac practically empty, i.e., free from any tension, so that the lung may be able gradually to expand.* It is a good plan each time the dressings are removed, to get the patient to cough once or twice (he often needs no prompting for this purpose), in order more thoroughly to evacuate any purulent matter that may remain. If the antiseptic method has been successfully carried out however, the amount of pus newly formed is very small. The drainage tube will require shortening as the cavity contracts and the lung expands, and will by degrees be removed altogether, when the wound rapidly heals.

I believe this to be the most successful method of treating recent cases of empyema, many cases having now been published which contrast very favourably with those in which other plans have been adopted.

Single opening and injection of pleura.—That the second plan, however, viz., that of making a free opening, evacuating the pus without any precautions to disinfect the air thus admitted into the pleura, and subsequently washing out the pleural cavity with disinfectants may prove successful, many examples might be cited to show. And moreover, it is not always possible to carry out rigorously the antiseptic plan. Hence I will relate the following case which will also afford me an opportunity of referring to some of the difficulties that may be met with in paracentesis, and the best means of avoiding them.

The case is that of a lady aged about 32, whom I saw in consultation with Dr. Charles Brown (now of Eastbourne) on January 27th, 1876. She had been taken ill ten days previously with

pains in the right chest, rusty expectoration, &c., and three days before the consultation, she had miscarried at the fifth month. The patient was lying on the right side much exhausted, with pulse 130, respirations 48 in the minute. The physical signs indicated pleuro-pneumonia on the right side, with a small amount of effusion. There was some diarrhoea present. The usual treatment was adopted, with an opiate for the diarrhoea, and a favourable prognosis was ventured.

On Feb. 4th, however, I again met Dr. Brown, the patient having the previous day had an attack of dyspnœa. She was sweating profusely, but stated that she had had no rigor; temperature between 101° and 102° , aspect pale and distressed, pulse very quick and feeble. The cough which had disappeared, returned with viscid and slightly tinged expectoration. The physical signs now were—dulness throughout the right side to mid sternum. Heart's apex in left axillary line. Respiration generally absent on the right side, except in the scapular region where some tubular breath-sound was heard, doubtless conducted from the left side, where, over the upper scapular region, the breathing was very tubular. Near the spine also on the right side some respiration was audible, obviously conducted from the opposite side.

It was evident that there was much effusion present, as to the nature of which the sweatings and the recent miscarriage caused some misgivings. Thoracentesis was mentioned as likely shortly to become necessary.

Feb. 6th. The symptoms having become more urgent, paracentesis was performed with the syphon apparatus in the manner described at page 232, and between 2 and 3 pints of sero-purulent fluid removed. Considerably more was left behind, but the urgent symptoms were immediately relieved.

We had the advantage of Mr. H. Arnott's help in these, and in the subsequent surgical proceedings. Great temporary relief was afforded by the operation, and, owing to the ex-

hausted state of the patient, more radical measures were not deemed expedient. Ten days later two more pints were removed, and on March 2nd, the fluid having again collected with threatening symptoms, it was decided to evacuate the whole of it under the antiseptic spray.

The necessary materials were at hand. A portion of the surface over the sixth space in axillary line having been frozen, an incision was made through the skin, and a full sized trochar thrust in. A considerable quantity of pus escaped, but unfortunately, the lower end of the spray-tube not being protected the tube suddenly became blocked and could not again be worked. Our antiseptic method having thus come to an untimely end, a drainage tube was inserted and secured by thread and strapping. The next day, however, symptoms of rapid re-accumulation appeared with profuse sweating, urgent dyspnœa, and great depression. The tube was withdrawn, and it was found to have become blocked by some inspissated pus; on its withdrawal a gush of thick slightly foetid pus took place, but there was no further escape although it was obvious that much more remained behind.

A bistoury was now introduced, guided by a director, deeply into the wound and the opening was enlarged one inch, but still only a small quantity of thick green pus came away. A piece of oiled lint was therefore inserted deeply in the wound, and carbolised tow applied externally. The patient who, naturally of nervous temperament, had borne these operations with great fortitude, was then ordered 3 grains of quinine and $\frac{1}{4}$ gr. of opium every three hours. During the night a sudden escape of a large quantity of pus took place from the wound giving her immediately a great sense of relief.

Let me pause here to make one or two remarks upon this difficult and instructive case at this stage of its treatment.

In the first place chloroform was deemed inadmissible, the lady having for some time previously complained of occasional fainting attacks attributed to "weak heart" and being much

exhausted by her present illness and recent miscarriage; and for similar reasons already stated, it was not thought expedient thoroughly to evacuate the pus at the second operation.

The unfortunate mishap with the spray-tube very forcibly illustrates the importance of protecting the tube by a linen filter. The tube was afterwards found to have been blocked at its point by a minute hair. The difficulties of the radical operation were in this case much increased by a very thick layer of adipose tissue which hindered all surgical proceedings. Whilst fluid could be withdrawn well enough by means of an ordinary trochar, even a large opening with a bistoury failed to be followed by a free flow. Subsequent reflection left little doubt that a false membranous layer was partially detached from the pleura, and that the opening through this membrane made by the bistoury or trochar did not, on the withdrawal of the instrument, accurately correspond with that in the intercostal space, and consequently a complete valve was formed; but that after a few hours the membrane softened and gave way before the pressure of the fluid. This very serious difficulty which may come in the way of the antiseptic method, would be best met by the employment of a large trochar and the introduction through the canula of the drainage tube over which the canula could be withdrawn. But it sometimes happens, especially in children, that the pleura is chambered by (false) membranous partitions. In such cases the only plan is to make a second opening through one of the posterior interspaces.

I will briefly relate the subsequent treatment of this case from a note with which Dr. Brown has favoured me.

A piece of gum elastic catheter, of medium size and four inches in length, was threaded an inch from one extremity by a piece of strong silk. The rest of the tube was inserted into the chest and secured there by means of the thread attached to pieces of strapping. A wooden peg was fitted to the end of the tube so as to serve as an effectual plug, and a soft diaper was the only dressing used.

The chest was washed out through the tube, at first every day, then every two or three days, as the symptoms and feeling of the patient demanded. For this purpose a wide mouthed bottle was obtained, into which one arm of a bent glass tube dipped deeply, the other being connected with two or three feet of tubing. The bottle was filled with the lotion to be used, (a weak solution of sulphate or chloride of zinc, or permanganate of potash, or quinine lotion,) and some of the lotion was drawn into the tube so as to fill it also. The peg was now removed from the catheter in the chest and the tubing attached when, on slightly raising the bottle, the lotion steadily flowed into the pleura: after allowing it to remain there a minute or two, it was again withdrawn, mingled with pus, by lowering the bottle to the floor and overflowed into a basin. As soon as the pleura had been so far as possible emptied, the plug was again inserted into the catheter and the patient left until next day when the process was repeated. The amount of fluid received by the pleura gradually diminished. It only became necessary to wash out the pleura every few days, the tube was gradually shortened and finally removed, and the wound healed in about three months from the time of the operation.

The patient took quinine and phosphate of iron tonic the whole of this time and slowly regained strength. I saw her early in the following August, when there was flattening and greatly restricted movement on the right side, but a considerable amount of resonance with weak vesicular breath sound to the third rib in front, and over the supra spinous fossa and scapular region. Some respiration was audible also below these points. The left lung was enlarged, extending across the median line. The heart sounds were feeble and distant. Measurements taken later in the autumn showed the right side to have a less circumference than the left by $2\frac{1}{8}$ inches. No symptoms were afterwards complained of beyond some drag-

ging pains felt occasionally in the right side, and a certain degree of shortness of breath during active exercise. Two years later this lady died from perforation of the bowel when apparently convalescent from a moderately severe attack of typhoid fever, during the course of which no chest symptoms presented themselves.

I have referred thus lengthily to this case, not that there was anything new in the treatment adopted, nor on account of its being ultimately successful, but because of the many points of practical importance in the treatment of empyema that it illustrates. Some of them have already been mentioned, of the others I may point out the method of washing out the pleura by means of the syphon, adopted and most ingeniously carried out by Dr. Brown, as the very best that can be employed. Here again the syphon has the advantage over the syringe, inasmuch as with it we know exactly how much force we are using, and this force is used equably and slowly. Amongst the sudden deaths that have occurred after paracentesis, not a few have happened whilst the pleura was being washed out. Syncope and cerebral embolism are the most common causes of such deaths; the one likely to be induced by any sudden increase or removal of intra-thoracic pressure, the other by such similar disturbance of pressure as may loosen any clots which may have formed in the veins of the compressed lung. Dr. Barlow and Mr. Parker³⁶ have suggested a good method of cleansing the pleura in children, viz., by placing them in a warm bath so that the openings on the affected side are submerged, some Condy's fluid being added to the bath water. The respiratory movements of the child would draw some of the water into the pleural openings, and thus wash out the pleura. This method would, however, probably only answer in cases in which there are two openings into the pleura.

Double opening and drainage tube.—In the event of our wishing to drain the pleura in the strict sense of the term, *i.e.*, by keeping in a tube which shall drain off the pus as rapidly as it forms, there is no method which we can fairly expect to succeed except that of making two openings, and passing a drainage tube through the chest from one to the other, the hinder of the two openings being at the lowest interspace we can select.

Dr. Goodhart, in the able paper in the “Guy’s Hospital Reports” above alluded to, shows very clearly that the attempt to keep the pleura drained by means of a syphon often ends in failure; out of 28 cases thus treated, ten died, and in only six cases did the syphon method alone suffice, other methods being afterwards adopted. The objection to the syphon method of draining the chest is, that the pleura cannot be drained of fluid by means of a syphon unless either the lung expands completely or air is freely allowed to enter the pleura. The first of these conditions is impossible in the cases under consideration, and the second equally so with a single opening and a single tube.

The double opening and drainage tube is not often employed as a primary measure, whether in acute or chronic cases of empyema. It is of course possible to pass a drainage tube through the pleural sac under the carbolised spray and to dress antiseptically, but it is difficult and inconvenient to do so. And as, provided the antiseptic method has been thoroughly carried out, we do not expect much subsequent suppuration, it would seem to entail unnecessary irritation and suffering to use such a means of drainage. The drainage may, however, be very usefully resorted to on the failure of one of the other methods. A long probe, or curved steel sound provided with a bulb at the end, is inserted either into the opening already present, or into a fresh one made at one of the anterior interspaces, and passed directly backwards and downwards so as to impinge posteriorly against one of the lowest interspaces. The surgeon then cuts down upon the bulbous end

of the sound, and having secured to it an india-rubber drainage tube, withdraws the sound, carrying the tubing with it through the chest. The two ends of the tube are then secured outside the chest in the usual way. By this means as fast as fluid forms in the pleura it is drained off.

In some cases in which we cannot hope for, or do not desire, the lung to expand, this is one of the best methods of treatment: but in all tolerably recent cases the expansion of the lung is one of the main objects of our solicitude and other methods are therefore preferable.

The treatment of *chronic empyema* of old-standing is a question of great difficulty, the cases which occur being so various in their nature and origin, and also in their duration and in the degree of expansibility of the lung. It is to be hoped that as time goes on, and effusions into the pleura are more promptly treated in the acute stages, these chronic cases will be less frequently met with. They may be divided into—

1. Cases of simple chronic empyema.
2. Cases in which the empyema is secondary to some lung disease, most commonly phthisis.
3. Cases in which the empyema has supervened upon pneumothorax.
4. Cases in which the empyema is associated with bronchial fistula or external sinus.

Then again the degree in which the fluid is purulent varies very much. In some cases it is greenish and opalescent, in others opaque creamy looking. In very few cases of old standing can the pus be regarded as active, or as any longer having, if I may so use the term, any malignant properties, the corpuscles being as a rule dead and more or less fattily changed. It is, however, much more difficult to deal with this purulent fluid than with serum, and active suppuration is also more readily set up in a pleura which has already yielded pus.

In these old-standing cases, we can no longer hope to gain

more than a very partial re-expansion of the compressed lung. Our treatment is directed to secure the removal of the fluid, and obliteration of the pleural cavity, the imperfect expansion of the lung being met by flattening of the chest-wall and enlargement of the opposite lung. Enlargement and increased function of the healthy lung is indeed the final aim of our treatment.

These results can only be obtained by prolonged and steady treatment. Two or three successive tappings with the trochar and syphon may be employed, with the object of withdrawing sufficient fluid to induce a negative pressure within the thorax, and thus to encourage expansion of lung, flattening of chest-wall, and encroachment of mediastinum. In the intervals of the tappings, the side should be strapped round its lower part, and a compress applied to the infra-mammary region, the part where flattening first commences. We may, by this means, cause the lung at its upper part to contract adhesions to the chest-wall, the gradual growth downward of which obliterates inch by inch the pleural cavity. The resonance of the opposite lung meanwhile gradually extends across the sternum, and the heart is drawn over with it.

Our degree of success in these cases depends mainly upon their duration. If, after a fair trial, we do not seem to be making further progress, a double opening and the insertion of a drainage tube is one plan left to us. Another is a free incision and complete evacuation of the purulent fluid under the carbolised spray, and the subsequent antiseptic treatment of the wound.

A case which I will now relate, however, induces me to think that it is still better after having, by one or two previous tappings, removed intra-thoracic pressure, to entirely evacuate the fluid under the antiseptic spray, to insert a tube for a few days only, and having thus let off any further fluid that may have collected to allow the wound to heal. Any fluid subsequently secreted *may* prove to be simply serous, and capable (with the aseptic air admitted) of being finally

absorbed. An experience of one case is not enough to do more than suggest this hopeful method of treatment.

The case also illustrates some other points in the treatment of purulent effusion secondary to phthisis or pneumothorax.

Daniel D., æt. 26, admitted into the Brompton Hospital under my care in May 1877.

There was nothing worthy of note in the early history of the patient, except that at the age of 18 years he had had some scrofulous abscesses in the neck, of which the scars still remained. Of dull phlegmatic temperament and vacant look, anything like a connected history of his chest illness was not to be obtained from him. But two and a half years previously, in September 1874, he had come under my observation as an out-patient, having for six months been complaining of cough and expectoration, with some pain in the left side and loss of flesh. My note at the time showed that there was a cavity at the apex of the left lung. He was admitted into the Hospital under the care of Dr. C. T. Williams, October 8th 1874, Dr. Williams' note then was:—"on the *right* side some scattered crepitation, mostly in front, with some dulness at the posterior base; on the *left* side, coarse crepitation over the upper third in front, with defective resonance over the lower half, and weak breath sound." The heart's apex was felt between the 4th and 5th ribs, about an inch to the left of the nipple. Dr. Williams surmised, chiefly from the position of the heart, that he had an old cavity at the left apex. He improved during his three months stay in the Hospital, gaining 12lb. in weight, viz., from 8st. 4lb., to 9st. 2lb., and the crepitant sounds on the right side diminished.⁸⁷

The patient continued under my observation, doing well until February 1875, after which time he began to fail, losing strength and suffering from pain in the left side. He ceased attendance at the Hospital, and I saw no more of him until his admission in May 1877.

⁸⁷ A brief summary of this case will be found at p. 261.

On re-admission into the Hospital under my care on May 8th 1877, the physical signs were of quite a different order.

The heart's impulse was perceptible between the right margin of the sternum and the right nipple. Right border of heart in nipple line. A slight systolic murmur was audible within the right nipple line. The left side of the chest was dull and toneless throughout, anteriorly and posteriorly, except at the sterno-clavicular region, where there was some tubular quality of the percussion note, and a certain degree of resonance in the supra-spinous fossa.

There was complete absence of respiratory murmur in front except immediately under the clavicle and in the sterno-clavicular region, where the respiration was weak and tubular, with slight dry crackle. Near the spine some respiratory murmur was audible (conducted from opposite side), elsewhere posteriorly respiration also absent. Spleen not perceptible. Diaphragm appears to act equally on the two sides.

Comparative measurements of the chest on the two sides, taken both above and below the nipple level, show $\frac{1}{4}$ inch excess on the left side. Extreme movement $\frac{1}{4}$ inch R. *nil* left. Temperature normal, pulse rather quick and feeble. Weight 8st. 10lbs. Nutrition of the body not good, nervous tremors of legs and straggling gait, so marked as to suggest ataxy. Scars of old abscesses in neck. Urine 1025, no albumen.

May 22nd. Paracentesis: 80 oz. of sero-pus removed by the syphon, in the manner described at page 232, the opportunity being taken of making some observations on the intra-thoracic pressure at different periods of the operation. (See annexed tracings and description, p. 264).

May 25th. Cough painful, dry. No elevation of temperature since operation. Heart's beat now imperceptible beyond right margin of sternum, its right border ascertained by percussion to be one inch to the right of the sternum. The apex-beat cannot be felt but is situated half an inch to left of the sternum. There is good resonance over the right side of the chest, but under the clavicle the respiration is weak, and

inspiration attended with fine spongy crepitation. On the left side (patient lying down) the percussion is very tubular under the clavicle to the 3rd rib. In the supra spinous fossa expiration is hollow, and some largish clicks are heard on cough.

May 29th. Evening temperature 97.5°, Pulse 75 R. 30. *30th.* T. 98.4°, P. 90, R. 25. *31st,* T. 97.4°.

June 1st. Cough dry, infrequent, no expectoration, no night sweats, no pain. Tongue clean. Appetite good. Evening Temperature 97.4°. *June 2nd,* Evening Temperature 98°. *4th,* Evening Temperature 97.8°.

June 8th. No cough. Heart's apex-beat, still half an inch to left of sternum. Some moist crackle with inspiration under the left clavicle. Weight 8st. 4lbs. *June 22nd,* Weight 8st. 8lbs.

July 6th. Weight 8st. 13lbs. No cough or expectoration. Feeling much better. Heart's impulse however, now to right of sternum, apex beat at ensiform cartilage. Some metallic clicks audible in left infra-clavicular region.

August 16th. Maximum impulse of heart, $\frac{3}{4}$ of an inch to left of *right* nipple line at nipple level. Impulse diffused from 3rd cartilage to this point. Left side dull throughout with some tubular quality of note in the clavicular and sub-clavicular regions, and some resonance in the supra spinous fossa. The dulness extends half an inch beyond the right border of the sternum. Complete absence of breath sound, vocal fremitus, and vocal resonance on the left side, except at the apex where, immediately under the clavicle, there is some cavernous respiration, and a few clicks are scattered down to the third rib. Voice sound conducted in supra spinous regions. Stomach resonance at ensiform level.

Right semi-circumference 17 inches, 2 inches above
nipple level.

Left	,	18	"	"
Right	,	16 $\frac{1}{4}$	„	1 $\frac{1}{4}$ inches below nipple level.
Left	,	17	„	"

The patient was now again suffering from the effects of intra-thoracic pressure, being breathless on slight effort.

August 17th. Paracentesis performed with M. Potain's aspirator, used in the manner indicated at page 237, and 76 oz. of sero-purulent non-foetid fluid removed. Unfortunately owing to one of the fittings accidentally slipping, a considerable quantity of air was admitted into the pleural cavity. The temperature however, notwithstanding this accident, remained at or below 98°, and the pulse between 60 and 80.

August 31st. Weight 8st. 12lbs.

Sept. 19th. Patient complains of pain in the left mammary region on breathing, and at noon to-day had a slight rigor. The temperature, however, at 3 p.m. was 98°, and in the evening 97·8°. An iodine application and a belladonna plaster removed the pain. He had no return of the rigor and continued to do well.

Oct. 26th. Left semi-circumference above nipple $17\frac{1}{4}$ inches.

Right	"	"	$17\frac{1}{4}$	"
Left	"	below	$16\frac{1}{4}$	"
Right	"	"	$16\frac{3}{4}$	"

shewing an equality of measurements above the nipple level, and a slight increase in favour of the right side below.

At this date there was a certain amount of resonance in front (patient standing) in the second space, below which the percussion note was dull. Sternum resonant. Vocal fremitus distinct over the supra-spinous and upper interscapular regions, none below the spine of the scapula. Slight vocal fremitus in front to second rib. Stomach note on percussion at fifth rib, nipple line. In the supra-spinous fossa crackle, increased after cough and marked bronchophony. Some respiratory murmur below the clavicle with slight crackle. Elsewhere silence. Heart behind the sternum. The patient has felt his breathing shorter during the past week. No cough nor expectoration, no pain. Tongue clean, appetite good, bowels regular.

Nov. 16th. Right $16\frac{3}{4}$ Left $16\frac{5}{8}$ below nipple level.
Right $17\frac{3}{4}$ Left $16\frac{1}{2}$ above nipple level.

Resonance of *right lung* extends to left margin of sternum as low as the third cartilage, thence sloping to right margin.

On the *left* side a certain degree of high-pitched resonance, somewhat tympanitic in quality, extended as low as the third rib: below this point dulness extending down to the fifth space nipple line and to the axilla: supra-spinous fossa, and to spine of scapula high pitched resonance, below dull. Supra-spinous fossa tubular cavernous breathing with scanty cough.

No breath-sound below spine of scapula. In front harsh muffled breath-sound to second rib, with prolonged expiration and a few dry crackles. No breath-sound below second rib. Vocal fremitus to second.

I may briefly sum up the main features of this case by saying that the patient, a young man of scrofulous habit, first came under my observation in September 1874, having been suffering from phthisical symptoms for six months. The physical signs then indicated the presence of a cavity at the apex of the left lung. He remained under treatment, doing well until February, 1875, when he began to lose strength and ceased attendance at the Hospital. On the 8th May, 1877, he was admitted under my care and the physical signs then showed extensive effusion into the left pleura, with great prostration of strength but no febrile disturbance. On the 22nd of May paracentesis was performed and 80 oz. of seropus removed. On August 17th the operation was repeated, 76 oz. being removed, and although on this occasion some air was accidentally admitted into the pleura, no evil results followed. The patient's general condition improved all this time, and he had had no cough or expectoration since his admission. But the physical signs showed that the pulmonary disease was only in abeyance. Towards the end of October for the first time, measurements showed that the right side was enlarging and the left diminishing in size, and percussion

and auscultation indicated expansion of the healthy right lung. A considerable quantity of fluid was still present however.

Dec. 5th. It was now decided after a consultation with my colleagues, Dr. Pollock and Dr. R. Thompson, to make an attempt more radically to cure the empyema; the object still in view being not to put any strain upon the left lung, which might reawaken the old disease in it, but to cause the gradual closure of the pleural sac by adhesion from above downwards.

A free opening was accordingly made in the eighth space a little in front of the posterior axillary line with a bistoury under the carbolised spray, the patient being under the influence of chloroform, and between two and three pints of purulent fluid were removed, air being freely admitted and the fluid evacuated as thoroughly as possible. A medium sized gutta-percha tube was then inserted and plugged, and the usual antiseptic dressings applied.

At the moment of free entry of air into the pleura a point of great interest was noticed, viz., that the heart became immediately displaced, and was seen to pulsate between the third and fifth cartilage to the right of the sternum.

The temperature was carefully observed for the next fortnight, and only on two or three occasions rose to 98.4° , being most generally below 98° .

Two days after the operation, on the tube being removed by Dr. Law, the Medical Officer who had performed the operation, a few drams only of *serous* fluid escaped from the pleura. The tube subsequently slipped, from some movements of the patient, and on attempting to introduce a fresh tube, it was found that the opening into the pleura had closed. Inasmuch as the fluid which had escaped from the pleura on the second day after the tapping was *serous*, and as there were no signs of any considerable re-collection, it was decided not to reopen the wound but to allow it to heal.

This decision has been so far justified by the result that there has been no reaccumulation of fluid. The heart's apex

is now (Jan. 10th) beating at the fifth space, just within the left nipple line. Stomach resonance is obtained at the sixth rib and the margin of the right lung, as judged by percussion resonance, and vesicular breath-sound, extends along the left edge of the sternum. The pleura contains some free air and probably a little fluid, but the pleural cavity is manifestly slowly contracting, and with its diminution in size the air and what little fluid there is—both at present indispensable to occupy what would otherwise be a vacuum—will, it may fairly be hoped, be gradually absorbed.³⁸

My object in relating this case is, firstly, to show that in the first instance a tentative treatment by several partial tappings at considerable intervals of time, and subsequently the more thorough evacuation of the fluid with the admission of carbolised air, is the best treatment for cases of empyema secondary to pulmonary disease. The liberality of the management of the Brompton Hospital, which permits of the lengthened stay in the Hospital of patients whose cases are of a kind likely to be permanently benefited thereby, should here be acknowledged. For I have no doubt that any attempt at once to deal radically with this case, would have again started active pulmonary disease.

Of course if the presence of hectic symptoms show the purulent fluid in the pleura to be active and noxious, it must at once be all removed, and in such cases the more freely air disinfected by the carbolised spray is admitted to take its place the better. But in the present case there were no such symptoms, and the fluid was only becoming harmful by its quantity giving rise to pressure symptoms, embarrassing the heart and the blood circulation, and impeding the respiratory movements of the other lung.

Again, secondly, this case is interesting, inasmuch as it

³⁸ This patient went subsequently to Eastbourne Convalescent Hospital and on his return a month later there was no re-collection of fluid, and he had no cough and was in good strength and flesh.

shows that a thorough evacuation of pus from the pleura under the antiseptic method introduced by Prof. Lister may, in some cases, lead to a complete change in the nature of the fluid subsequently effused. And that possibly the best method of dealing with future cases of empyema, will be found to be to keep the opening patent for a few days only, and to allow it after that period to close, awaiting the result. If only serous fluid is secondarily secreted, it will probably be absorbed without difficulty. If not, the same operation, or aspiration, can be repeated at any future time.

In acute cases of empyema, in which we have reason to believe that the suppurative process has not entirely ceased, the wound may be kept open and the nature of the fluid observed for a longer period.

TRACINGS SHOWING INTRA-THORACIC PRESSURE DURING PARACENTESIS.

FIG. 13.

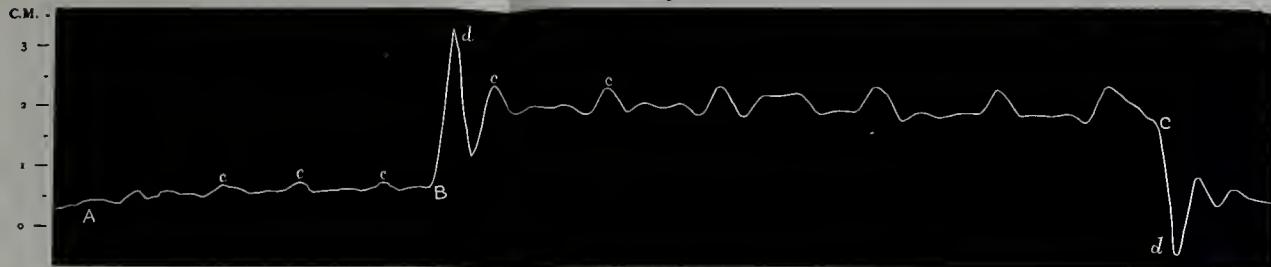
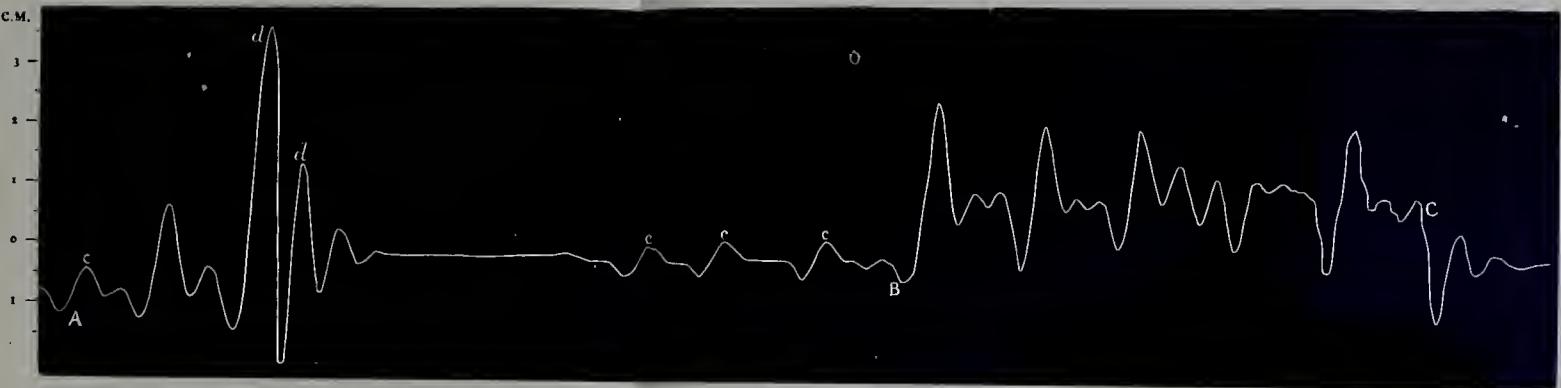


FIG. 14.



The above tracings show the intra-thoracic pressure at different periods of the operation performed on May 22nd. They were obtained by attaching to the syphon tube a side branch connected with a manometer so placed that the surface of the mercury was on the same level as the trochar in the chest.* By means of a float resting upon the surface of the mercury and having a light whalebone index attached to it, the movements of the mercury were recorded upon a piece of smoked paper attached to a revolving drum. The drum was timed to make a single revolution in a minute.

The tracings, of which the annexed figures are but small sections, are difficult to interpret in all their details; indeed they are not so easy to take as one might imagine, but after an experience of three cases in which they have been taken in a similar way, I regard these as accurate so far as they go, and therefore as worthy of being recorded to compare with others.

Tracing No. 1, fig. 13, is a portion of that taken at the commencement of the operation. The first portion A, B, was taken whilst the fluid was escaping through the syphon, the fall both from the puncture in the chest and from the mercurial level being 2 feet of water. At B, the exit tube was pinched so as to arrest the flow, and there is an immediate rise of mercurial pressure equal to 19 centimetre during expiration, 12 c.m. during inspiration. At this period (the commencement) of the operation the mercurial columns did not become level, i.e., there was still some slight positive pressure, whilst the

fluid was escaping, so that these figures represent a degree of pressure rather below that really present within the thorax; the real numbers being as indicated by the scale 2·5 c.m. during expiration, and 2 c.m. during inspiration, mean pressure 2·25 c.m.; c c c represent respiratory movements, d d are larger undulations due to sudden alteration in the mercurial pressure on closing and opening the exit tube respectively.

Tracing 2, fig. 14, is from that taken towards the close of the operation. In the first portion A, B, whilst the fluid was still flowing freely it may be noticed that the respiratory waves c c c c are larger. The mercurial surfaces were now nearly level whilst the syphon was in action, and shewed negative pressure during inspiration, in fact, as will be seen by the tracing, they oscillated pretty evenly during respiration: d, d, are larger undulations produced by coughing. At B the exit tube was again pinched below the branch connected with the manometer, and the pressure is now seen to be 2 c.m. in expiration, the mathematical sign - (minus) - 5 c.m. to 0 in inspiration, mean 7·5 c.m.

A certain deduction should however be made from these figures on account of the slight syphon (negative) pressure in action previous to the moment at which the flow was arrested at B. I must add also that there is some uncertainty as to the exact degree of this negative pressure present, whilst the syphon was acting, which makes a slight error possible in the position of the scale attached to this latter tracing. The base line was readily enough obtained for the first tracing, but it could not be relied upon for the later ones. This is a point to be attended to in future tracings of the kind.

* In the manner suggested in a paper read before the Clinical Society, Jan. 1870.
Trans. Vol. III.

CHAPTER VI.

ON FALSE OR SPURIOUS HÆMOPTYSIS.

HÆMORRHAGE may arise:—1, from the nasal membrane; 2, from ulcerated throat; 3, from intentional injury; 4, from the gums and alveoli; 5, from a scorbutic condition of the mucous membranes; 6, from *diapedesis* in anaemia; 7, from more copious mucous haemorrhage in hæmophilia.

By true hæmoptysis is meant haemorrhage from the lungs, either from the lung texture proper, or from the lining of the bronchial tubes ramifying through the lungs.

By false or spurious hæmoptysis is meant the spitting of blood which has escaped from some portion of the mucous membrane lining the nasal or buccal or pharyngeal passages. Perhaps the true anatomical line of division between true and false hæmoptysis would be at the glottis, for below this point the mucous membrane assumes the ciliated columnar epithelium characteristic of the bronchial tract, whilst above the epithelium is of the squamous kind. In true hæmoptysis, with the exception of those very rare cases in which the haemorrhage comes from the trachea, the blood escapes from the pulmonary or bronchial vessels; in false hæmoptysis from branches of the carotid trunks. The parts where the blood of false hæmoptysis is derived are, the nasal mucous membrane, the pharynx, and the gums and dental alveoli.

i. In cases of decided epistaxis the blood commonly trickles down the back of the throat, and excites cough, by which it is removed in clots and mixed with saliva: the source of haemorrhage is obviously, however, the nasal membrane, and no real difficulty in diagnosis ever arises. It is only in cases in which the nasal haemorrhage is but slight, and attended with little or no escape of blood through the anterior nares, that there is any probability of the affection being mistaken for hæmoptysis. This occurrence may happen at night, and

the patient wake up spitting blood. The absence of all cough and chest signs during the day, the absence of fever, and the signs of blood mingled with the nasal mucus when expelled, or the discovery of some coagula in the nasal passages, will render the diagnosis in these cases also clear.

2. Ulceration of the throat may lead to copious hæmorrhage, and in these cases again, no difficulty is likely to arise in the way of diagnosis.

3. A class of cases is now and again met with which occasions much trouble to the practitioner, and requires much decision in management. These are cases of feigned or hysterical hæmoptysis. Unfortunately, it is too much the custom to regard all cases of spurious hæmoptysis as hysterical, a term for which there is in the majority of instances no possible foundation. For hysterical hæmoptysis is nothing more nor less than downright attempt at imposition, the blood being produced either by sucking the gums, or by pricking or incising them. Dr. Johnson⁸⁹ has referred to a case of a young girl sent up to King's College Hospital by a lady interested in her, with an elaborate history of symptoms, including blood-spitting. The character of the expectoration which consisted of unaërated saliva mixed with fresh blood, was sufficient to indicate its source, and on examining the mouth with a bright light, about twenty fine cuts or scratches were discovered on the mucous membrane covering the hard palate. A sharp reprimand and a short course of shower baths and steel tonics, speedily removed the symptoms.

In some hysterical cases, however, I have known the blood-stained saliva to be highly aërated, probably produced by sucking the gums after injuring them with a needle. The appearance and physionomy of the patient is generally sufficient to excite suspicion, and other hysterical symptoms are usually present.

4. A morbid state of the gums frequently arises from want

⁸⁹ *Medical Times and Gazette*, April, 1862.

of due attention to the teeth, or from the presence of decayed stumps in the alveoli. On the slightest touch or friction blood exudes from the mucous membrane, which is swollen and congested, a livid line running along the margin of the gum. A similar condition arises from the effects of certain drugs, especially mercury, and, to a much less degree, lead and iodide of potassium.

In these cases there is fœtor of breath, and an inspection of the gums and teeth at once suggests the probable source of the blood-spitting, which is usually insignificant in amount and unaccompanied by any cough or chest symptoms.

The treatment of these cases falls partly within the province of the dentist: it consists of the employment of astringent tooth-powders, of which one of the best for the purpose consists of finely-powdered kino one part, to three or five of prepared chalk, with or without a little animal charcoal. Any decayed teeth must be removed or stopped. More or less dyspepsia is usually present in these cases, partly arising no doubt from the condition of the gums and teeth, and a stomach-cough added to the staining of the sputa may suggest to the patient that he is consumptive.

5. An insufficient supply of vegetable food, a very common dietary error among all classes of people in towns, leads to a spongy congested state of the mucous membrane of the mouth and fauces, of the same kind as that which, in a more intense degree, is associated with the other lesions characteristic of scurvy. This is one of the most common causes of spurious hæmoptysis. The relaxed condition of the throat, resulting in the secretion of an undue amount of viscid mucus, gives rise to some cough, and the mucus expectorated or rather hawked up from the pharynx, together with the saliva from the mouth, is from time to time tinged with blood. It is often very difficult to distinguish this form of false from true hæmoptysis. And indeed the condition of the mouth is but a sample of that of the mucous membranes

generally; and the larger bronchi if affected with catarrh, are apt to yield a viscid and slightly stained secretion.

I have, moreover, observed in some cases of phthisis a staining of the expectoration which has seemed to me to have arisen in the same way. Anything like considerable hæmoptysis does not, however, arise from this cause, but rather such a staining of the sputa as may suggest fresh congestion or pneumonia.

In the cases under consideration, however, there are no pulmonary signs to be discovered on a very careful examination. The hæmorrhage is never in quantity, it consists of a tinging or streaking of sputa which is distinctly made up of mucus mixed with saliva, giving rise to a dirty red mixture containing some little streaks or clots of blood. On microscopic examination squamous epithelium cells are seen in abundance, and red blood corpuscles are but thinly scattered over the field. The patient complains of the taste of blood in the mouth, and this is especially disagreeable after sleep. The nutrition is not good, the muscles are flabby and wanting in tone, and the patient feels languid and out of sorts. There is commonly some anæmia present.

A most favourable prognosis may confidently be given in these cases, if we are quite satisfied as to the absence of any pulmonary sign.

In their *treatment* the diet must be attended to, an abundance of fresh fruit and vegetables being added. Five or ten grains of citrate of iron should be ordered in fresh lemon juice two or three times a day, and cod-liver oil may often be given with great advantage. Some tannin solution should be used night and morning as a gargle, and to rinse the mouth.

In cases of phthisis in which we suspect this morbid condition of the bronchial mucous membrane to be present, fresh lemon juice, a not unpleasant vehicle for cod-liver oil, is very valuable.

6. In certain cases of anæmia, cases attended with all the

other phenomena of that disease, the mucous membrane of the mouth and fauces, although pallid in appearance, exudes a sanguineous fluid, which, mixed with the saliva, causes spurious hæmoptysis. There is in these cases, so far as one can discover no true haemorrhage from the membrane, no bleeding points can be seen, but in the course of twenty-four hours a considerable amount of blood will transude through the vessels. The transudation is ordinarily very slow, and in the day time is scarcely noticed, but during the night some accumulation takes place, and on waking the patient expels perhaps an ounce or more of bright red unaërated fluid containing a few coagulated films, giving an appearance closely resembling that of currant jelly and water. Some of the sanguineous fluid often escapes from the mouth upon the pillow during sleep. The patients suffering from this affection are mostly females: amongst other symptoms of anaëmia the menstruation is disordered or suppressed, and commonly, but not always, at the menstrual period the escape of blood from the mouth is considerably increased. Probably from the same cause, *i.e.*, an increased blood-pressure finding relief at the surface of least resistance, any extra exertion is apt to cause an increase in the sanguineous flow. The real pathology of these cases, is however, confessedly obscure: but this is the variety of false hæmoptysis for which we are most often consulted. The patients are breathless, they sometimes have a hard cough, and complain of pain in the left side and considerable prostration, which symptoms, with increasing pallor and blood-stained expectoration, are quite sufficient to persuade them and their friends that they are consumptive.

In such cases again, the greatest pains must be taken to come to an accurate physical diagnosis. The respiratory sounds will usually be found to be weak and partly suppressed from want of muscular power. The percussion is, however, even on the two sides, and the respiration although feeble is

vesicular. The character of the cough, both as heard through the stethoscope and otherwise, is usually quite distinct from that of chest disease. Then, there are all the physical signs of anaemia present, venous hum, arterial murmurs, &c. and the "pain in the chest" is without much difficulty ascertained to be either infra-mammary neuralgia or gastrodynia. Some patients with this form of spurious hæmoptysis have plenty of colour in the cheeks and are plump rather than emaciated, but they nevertheless present the physical signs of anaemia. The careful observation of a large number of these cases for long periods, enables me to say that it is most rare for them to become phthisical.

The condition calling for *treatment* is the anaemia. More fresh meat must be taken, and if necessary, some pepsine added to aid digestion. In some cases there is considerable disorder of stomach present which must be first set right before the remedies appropriate to anaemia can be given. These remedies are the astringent forms of iron. Cold salt baths or sea-bathing allowed only for a very brief time, one or two minutes, and immediately followed by vigorous frictions are extremely useful in the convalescent stage. Abundance of fresh air and out-of-door exercise is of course to be insisted upon. The patients and their friends are often much afraid of fresh air, and the cases have usually at the period at which they come under observation been aggravated by confinement in warm and ill-ventilated rooms. It often happens that there are decayed teeth present, setting up irritation in the gums and increasing the disposition to haemorrhage. We must be cautious, however, about advising such to be at once removed, for in these patients the blood is peculiarly aplastic, and haemorrhage difficult to control.

7. General haemorrhage from the whole mucous membrane of the mouth is sometimes seen in haemophilia. Although doubtless differing chiefly in degree from that described under the preceding heading, there being no perceptible lesion dis-

coverable in the mucous membrane, this hæmorrhage is usually so considerable in amount as to prevent any possibility of mistaking its nature. A more or less strongly marked predisposition to hæmophilia is usually also present, and other signs of the disease may be found.

CHAPTER VII.

THE RESPIRATORY MECHANISM IN HEALTH AND DISEASE.

MECHANISM by which first expansion of lungs effected—Elasticity of lungs not entirely relaxed at end of expiration—How lungs maintained in the semi-expanded state—Residual tension of lungs—Use of schema to illustrate mechanism of respiration—Explanation of conditions present in Emphysema, Pneumonia, Pleuritic Effusion and Pneumothorax, by means of the schema—Some disease effects of atmospheric pressure.

IN concluding this work, I will venture to make a few remarks upon the mechanism of respiration, and its modifications in disease; remarks which will have found clinical application in many of the preceding pages.

The subject is one not only of great interest in itself as unfolding to our contemplation some of those minor adaptations of living mechanism which no art can effect or imitate, but of the greatest importance in leading us rightly to appreciate the results of disease which are mechanically induced, as well as those which are dependent upon vital changes.

In the new-born child, on the placental circulation being interrupted, carbonic acid accumulates in the blood and stimulates the respiratory nervous centres, thus exciting acts of inspiration. This central nervous stimulation would probably not alone be sufficient, but it is supplemented on the first exposure of the child to the external air, by a general excitation of the cutaneous nerves causing strong inspiratory movements.⁴⁰ In the Gulstonian Lectures for 1872, Dr. Hensley conjectured that the first expansion or unfolding of the air-cells of the lungs in the infant, was caused by the penetration of the blood through their capillaries: and this view was supported by some experiments made in the same year by Liebermann. Liebermann roughly imitated the

⁴⁰ See Pflüger's experiments referred to in Carpenter's *Physiology*, 8th edit., p. 387.

structure of an air-sac by taking two ox-bladders, placing one within the other, and including between them a layer of india-rubber tubing. On injecting oil through the tubing the bag, previously collapsed, expanded drawing in air with a perceptible sound.⁴¹ Probably all these forces come into operation nearly simultaneously, and we thus get successive inspiratory expansions of the chest, in obedience to which the air penetrates the recesses of the lungs, and is never again fully expelled.

It is not at all curious that the lungs, thus once inflated, should remain more or less permanently expanded, retaining in their interior a certain amount of air. They, like any ordinary elastic air bags, would do this, whether in or out of the body. But in the body, whether during life or after death (within the limits of ordinary respiration) the lungs are expanded *beyond* the point to which they would, from their mere elasticity, revert. This is a fact admitted by modern physiologists, but not, so far as I am aware, explained by them, nor is its importance in clinical medicine sufficiently recognised. I am therefore the more desirous to draw attention to it here.

We have no difficulty in understanding how, after the separation of the placenta, the gradual accumulation of carbonic acid should excite the pneumogastric centres to initiate respiratory movements, and how further excitation should be conveyed to these centres from the peripheral nerves, on the sudden contact of cool air with the surface of the body. Moreover, we may well believe that the physical effect of a diversion of the blood through the lungs would be to unfold the air vesicles, and thus to enable them to become permeated with air. In order to account, however, for the maintenance of the lung in the semi-expanded state we must further assume

⁴¹ *Wien. Med. Zeit.* No. 5, 1872. I am indebted to Dr. Lauder Brunton for the reference to this experiment. Hensley's lecture is briefly alluded to in the *Brit. Med. Journal*, June, 1872.

that once the muscles of respiration, which are for the most part inspiratory muscles, have been excited to action, they remain permanently shortened by virtue of their vital contractility or *tonus*. And in the course of tissue growth the ribs and cartilages become moulded to that wider arc which they have thus been brought to assume.

The contractile power of the lungs derived from their residual tension, has been measured by different observers with somewhat different results. Dr. Carson in 1820,⁴² demonstrated the existence of this reserve tension and estimated it in different animals as equivalent to from six to twenty or more inches of water, according to the size of the animal. He fully perceived the importance of this elastic force, both in the respiratory mechanism and as an aid to the circulation. Dr. Carson indeed regarded the respiratory movements as due to an antagonism between the elastic resilience of the lungs, and the muscular action of the diaphragm. He maintained that the traction of the lungs upon the diaphragm gave rise to a sense of uneasiness, to overcome which the muscle contracted, but that the enduring lung elasticity on the cessation of the muscular effort again drew up the diaphragm. This view although erroneous was based upon an accurate observation of phenomena which were for long afterwards ignored.

Donders in 1853,⁴³ made some experiments on the human subject, connecting a manometer with the trachea, and then carefully opening the thorax. In this way he ascertained the residual elastic tension of the lungs, and concluded that in the healthy person it was equal to 80 millimeters (about 3 inches) of water. He further added 20 mm. for the tonicity of contractile elements in the lung.

Dr. Salter in 1865⁴⁴ found the residual tension in the dog equal to 4 inches of water.

⁴² *Philosophical Transactions*, 1820, pt. i, p. 29.

⁴³ *Leitschrift für Rationelle Medicin*, 1853, p. 290.

⁴⁴ Lectures at the College of Physicians, *Lancet*, 1865, vol. ii, p. 142.

Dr. M. Perls in 1869⁴⁵ gives the result of one hundred experiments, conducted in the same manner as Donders, upon persons who had died of different diseases: out of these, in 25 cases death had been caused by diseases remote from the lungs, although in most instances the lungs were in some degree affected. The highest degree of residual tension registered by Dr. Perls' manometer from these 25 cases was 60 mm. of water, the lowest 5 mm.: mean 35·3 mm.

It is very possible that the experiments of both Donders and Perls give results somewhat short of those of perfect health. It is, at all events, singular that the highest elastic pressure obtained by Perls from one of the 25 cases in which the lungs were presumably healthy, does not equal that (63 mm.) obtained from a case of bronchitis and one of phthisis.

The amount of muscular tissue in the human lung is at most but very scanty. Donders added 20 mm. to his estimate of residual lung contractility on account of muscular tonicity. Stricker admits that here and there in the free margins of the alveolar passages nearest the bronchial terminations from which they are prolonged, delicate bands of smooth muscular fibre are to be found "which often consist of merely an isolated fibre imbedded in a delicate connective tissue. The membranous walls of the alveoli themselves are entirely destitute of muscular tissue, nor have I been able to discover any muscular fibres in the more compact borders of the individual alveolar septa."⁴⁶ It seems that this tissue is more abundant in the lungs of animals. Muller found the lung of a dog contract on the application of iced water.⁴⁷

Although in the human subject we may conclude that so far as the lung proper is concerned, muscular action takes no

⁴⁵ *Deutsches Archiv für Klinische Medicin*, July, 1869.

⁴⁶ *Human and Comparative Histology*, Syd. Soc. Edit., vol. ii, p. 60.

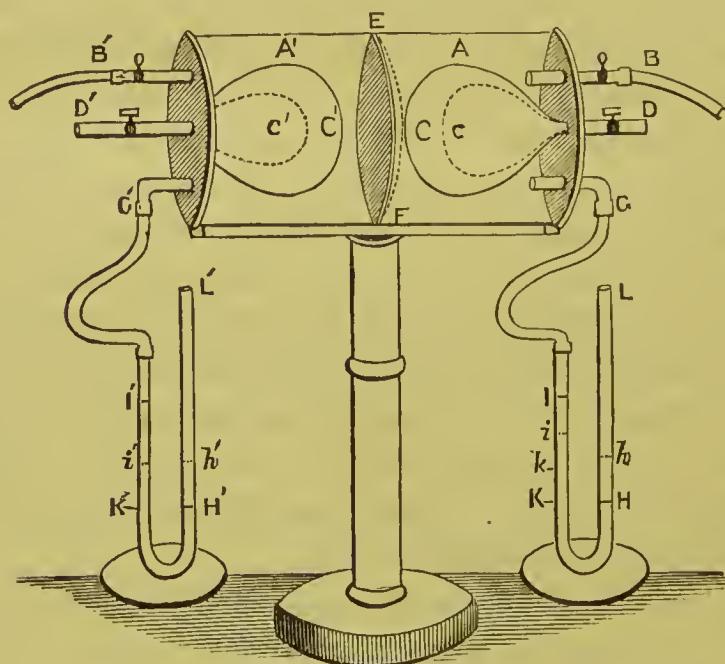
⁴⁷ Ludwig's *Arbeiten*, 1869, p. 64. Dr. Brunton informs me that this contractility was found to continue for two days if the lungs were supplied with blood by artificial circulation.

direct part in the respiratory mechanism, it is yet impossible not to see that the muscular bronchial tubes ramifying throughout the lungs with whose texture they are in intimate union, must indirectly add to their contractility. We are more concerned now, however, with the purely elastic elements of the breathing apparatus.

The schema represented in Fig. 15, was designed for me in December, 1876, to illustrate some points connected with healthy and diseased respiration, referred to in the course of a clinical lecture delivered at the Brompton Hospital.⁴⁸

The object of the schema—a plan or diagram-model of the chest—is to show the main physical conditions present in the chest, and how they are modified (*a*) during normal respiratory movements (*b*) in certain diseases.

FIG. 15.



The schema consists of a cylinder of glass, closed at each end by a metal plate screwed on and perforated for the admission of certain tubes. A central partition, EF, made of sheet india-rubber, divides the cylinder into two air-tight

⁴⁸ On excavation of the lungs in Phthisis. Lec. vi—*Lancet*, Jan., 1878.

compartments, each of which, as will be presently seen, represents one half of the thorax.

Tube D represents the trachea, and is connected with an elastic bag, ϵ , representing the (left) lung. Tube G communicates with the space between the lung and the wall of the chamber, which space, therefore, corresponds to the pleural cavity. This tube is connected with a mercurial manometer, the free end of which, L, is open to the normal atmospheric pressure. Tube B also communicates with the same (pleural) space, and is provided with a stopcock and a mouth-piece.

Exactly the same parts are repeated on the opposite side of the partition, EF, which therefore represents the mediastinum.

The apparatus must be ascertained to be thoroughly airtight. Then by partially exhausting the air from chamber A through tube B (trachea tubes D and D' remaining open) we cause the bag ϵ to expand (C), the mercury in the manometer (H K I) to rise towards the chamber, and the mediastinum EF to become convex, as indicated by the dotted line. By closing the stopcock B we maintain all the parts in this position.

If, next, we repeat the same process on the opposite side—partially exhausting chamber A' through B' until the mercury I' is at the same level as I; the bag ϵ' will expand to C', the mediastinum, E F again become vertical, and by closing stopcock B' the parts will be maintained in this position of equilibrium on the two sides.

In the schema thus arranged, we have the conditions of the healthy chest rudely but accurately imitated. The two chambers represent the two sides of the chest, each containing a semi-expanded lung C, C', surrounded by a pleural cavity A, A', (here greatly exaggerated,⁴⁹ the cavity being rather poten-

⁴⁹ This is unavoidable since the walls of the schema are rigid, and if the bags fitted accurately, their further expansion in inspiration could not be represented. No fallacy is hereby introduced however.

tial than real in the healthy chest), each cavity being separated from that on the opposite side by the mediastinum E F, which is common to both and equipoised between them.

The wall of the natural thorax is, however, elastic or resilient in every part, although much more stiffly so than the lungs. We cannot exactly represent this resilience of the thoracic walls in our schema. The only parts of our apparatus which are at liberty to yield to the excess of external atmospheric pressure over that within the pleuræ, are the small surfaces of mercury at H H'. Hence the elevation of the mercury K I, K' I' towards the chamber on each side, multiplied by the area of apertures G G', and divided by that of the whole surface of the chamber, would represent in millimeters the amount of recession of each portion of the thorax, provided each portion were equally resilient.

Although in the natural chest, the diaphragm yields far more than any other portion of the chest wall to the traction of the lungs, this is chiefly because its resistance is weaker, so that a recession of an inch on the part of the diaphragm is only equivalent to the recession of one or two millemeters on the part of the ribs or cartilages. For it must be further remembered, that the diaphragm is not elastic, that the limits of its *tension* are therefore very narrow; and indeed, the elastic recoil of the diaphragm depends mainly upon the spring of the cartilages to which it is attached. Our mercurial columns therefore, after all, very conveniently and fairly represent the whole thoracic resilience in a lump sum.

Having thus with the aid of our schema rehearsed the statical conditions of the chest, the dynamics of respiration may be easily demonstrated.

In order to imitate an inspiration, aspiration must be made simultaneously through the tubes L and L', thus representing the contraction of the diaphragm and inspiratory muscles on the two sides.

As the mercurial columns rise up the limbs L, L' of the

manometers, representing the expansion of the thorax, the lungs C C' enlarge by the entry of air through trachea tubes D and D'.

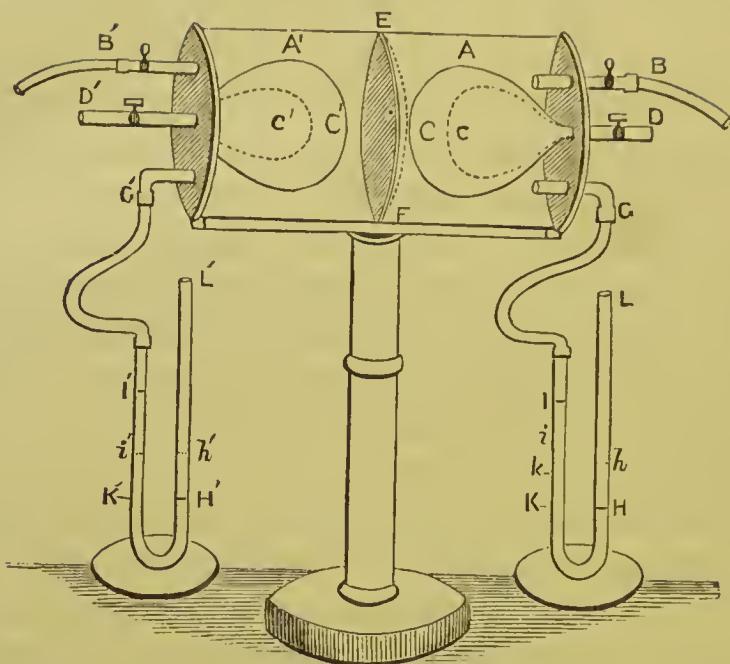
It will be observed that during the first part at least of inspiration, the weight of the two columns of mercury K I, K' I' tells in favour of the inspiration. This weight of mercury corresponds as before said to the outward resilience of the thoracic walls, and counter-balances the elastic traction of the lungs. This is a fact, well shown in the schema, which it is important to bear in mind. It may be stated in the proposition that, *in health the resilience of the chest wall is in favour of inspiration.*

From some observations on the dead subject which I made two years ago, I am inclined to think that this elastic aid to inspiration obtains throughout the act in calm breathing. Respiration is thus rendered smoother and less laborious, elasticity entering as an important item into the inspiratory, as it has been long known to do into the expiratory act.

The conditions present in the chest as shown by the schema must be further examined in their effects upon the heart and circulation.

The disposition to the formation of a vacuum in the intrapleural space A, equal to the weight of the column of mercury K I, causes an aspiration towards that cavity which was at first shewn by the convexity of the mediastinum E F (dotted line): and there being a similar and equal aspiration towards the pleural cavity on the opposite side of the mediastinum, it follows that there is a constant determination of blood towards the cavities and walls of the heart—a hollow organ situated within the mediastinum, and communicating by a system of tubes with parts outside the thorax. This central attraction is forcibly overcome by the muscular contraction of the heart but resumes its sway at the termination of systole, aiding the return of blood to the flaccid heart cavities, and encouraging the flow through the coronary vessels. This aspiration towards

FIG. 15.



the heart is, be it remembered, in health a constant force, increased during inspiration, held in momentary abeyance during the more forcible muscular contraction of the heart, but not wholly extinguished even at the end of ordinary expiration.

Let me now very briefly refer to a few chest diseases considered in their mechanical relations to these normal conditions.

1. *Emphysema*.—If we imitate the effects of repeated coughing upon the “lungs” in our schema, so as to partially *wear out* their elasticity, they will become larger so as to occupy more of the thoracic space, and *pari passu* with the loss of elasticity will the mercurial surfaces of the manometers become more and more level. In other words, as the lungs become relaxed, the thoracic resilience dominates, and the chest enlarges, *i.e.*, the ribs and cartilages expand, and the diaphragm flattens, (page 186).

If, in the lapse of time, any molecular change occurs in the material composing the bags A A' so as to relax their elasticity, the same effect will be produced, the columns of mercury representing thoracic resilience will gradually become more

and more level, the phenomena of degenerative emphysema being thus imitated, (page 191).

So long as the tracheal tubes D, D' remain open, however, it is impossible for the surfaces of the mercury columns I K, I'K' to be depressed to a lower level than H H'; and I believe it is likewise impossible for the thoracic wall or diaphragm in emphysema to be displaced by any *pressure* exercised by the enlarged lungs.

Although no direct pressure is exercised upon the heart, however, the annihilation of the aspiration effects of the lungs upon its cavities and walls must in extreme emphysema be a material loss in the mechanism of the circulation, and by impeding the coronary circulation, must impair the nutrition of the heart itself.

Again, in extreme cases, the complete extinction of the excentric resilience of the thorax as an aid to inspiration renders this act much more laborious, because entirely muscular, and the absence of any reserve elasticity in the lungs renders the greater portion of expiration also voluntary. (pp. 16, 186).

A glance at the Schema shows at once that any general alteration in the tenuity or denseness of the air cannot affect the mechanical conditions present in emphysema, but any rarefaction of air within the lungs (as by expiration into a rarefied air) would powerfully aid their contraction. Page 197.

2. *Pneumonia*.—The engorged and consolidated lung in pneumonia is considerably enlarged, *i.e.*, its mean bulk is considerably greater than that of the opposite lung. How is it that a somewhat enlarged solidified organ is not compressed by the thoracic wall?

The answer will perhaps be given that it *is* compressed. It is uncommon, however, to find the impression of the ribs upon the surface of the consolidated lung in pneumonia; the exudation upon the pleura is finely granular, showing rather

the reverse of pressure; the intense bronchial breathing, and abundant *inspiratory* crepitation are also against the idea of pressure. Finally, the pain in pneumonia is not of that severe continuous character that compression of an inflamed organ (*e.g.*, the testicle) gives. The reserve capacity of the thoracic walls and diaphragm to increase the dimensions of the thorax synchronously with the enlargement of the contained viscera, is a beautiful means of adaptation to the frequent temporary exigencies of the lungs, and in pneumonia serves to relieve the tender organ from all pressure.

3. *Pleuritic Effusion.*—(a). Fluid effusion. Very little need be added to what has already been said respecting the mechanical conditions present in fluid effusions. If the end of tube B' of the schema be dipped under water, and the stop-cock opened, the water will flow into the chamber (pleura) A' so long as there is any unrelaxed elasticity in the bag C', the mercurial column I' K' will become lower and lower, until it is level with H'. But, more than this, the bag C in the opposite chamber will partially contract, the mercurial column I K, become shorter, and the mediastinum E F, become convex towards A. After the mercurial columns of manometer L' become level it is needless to say that no more water will be drawn into the chamber, A'.

Here are exactly imitated the conditions present in moderate effusion into the pleura, *i.e.*, gradual yielding outwards of the thorax, slow descent of the diaphragm, and displacement of the heart as the fluid accumulates; yet neither of these phenomena are due to the pressure of the fluid, but all to the relaxed lung gradually releasing the tension of the opposite lung, and of the thoracic parieties.

There seems to be some difference in the opinion of authorities as to the size to which the human lung will collapse by virtue of its elasticity. Dr. G. M. Garland quotes Rokitansky to the effect that the lung will contract to one eighth of its usual volume, but Dr. Sharpey's estimate is probably nearer

the mark, viz., that when the thorax is opened, the lungs will contract to about one third of their bulk.⁵⁰ To this point, then, an effusion may encroach upon a previously healthy lung without exciting any pressure upon it.⁵¹ Garland⁵² has performed the experiment just described in the schema, with the pleura of a dog, and the mere elasticity of the lung drew up "a large body of fluid into the chest" through a tube immersed in fluid at a slightly lower level. The same author has made numerous experiments to show that even in considerable effusions the diaphragm remains arched, supported in that position by the retractility of the lung "until the weight of the fluid exceeds the lifting force of the lung." In his experiments Dr. Garland does not take into sufficient account the thoracic and diaphragmatic resilience represented in the schema by the mercurial columns I K, I' K'. But the diaphragm will not descend far by virtue of its resilience alone, probably only a few millimeters; hence, as Garland's models show, and as Traube clinically points out, the diaphragm remains arched even in considerable effusions. This point cannot be shown by our schema. It is very different, however, with the heart. The moment equilibrium between the two lungs is destroyed by the relaxation of the elasticity of one of them, it is restored by the partial contraction of the opposite lung, and the shifting with it of the mediastinum and heart. Hence, displacement of the heart in liquid effusions is (the lungs being sound) directly in proportion to the effusion.

If the water vessel in which B' is supposed to be immersed be raised, the chamber A' becomes injected with fluid at a certain pressure. The bag c' at once begins to collapse from pressure, the mediastinum to be still more displaced, and the mercurial column rises towards L', indicating the pressure employed. The opposite bag c recedes before the advancing fluid, and the mercurial columns on that side become level.

⁵⁰ Quain's *Anatomy*, 6th edit., vol. iii, p. 274.

⁵¹ loc. cit., p. 61.

⁵² p. 52.

These are the obvious effects of pressure similar to those of excessive fluid effusion into one pleura. The illustration in the schema of what happens on the healthy side as the fluid advances, how the thoracic walls expand to the limit of their resilience making way for the heart, and relieving it from pressure to the latest moment, is interesting, and a true diagram of what occurs in nature. The late appearance of any increase in the comparative measurement of the chest on the side of the effusion is thus accounted for. There is at first an increase in the *total* circumference, and only in extreme effusions is there comparative increase on the affected side. Increase of measurement on the affected side, therefore is, other causes being excluded, an evidence of positive intra-thoracic pressure. Bulging downwards of the diaphragm has the same significance.

Displacement of heart *per se* is not significant of pressure.

b. Pneumothorax. The effect of admitting air into the pleura is somewhat different from that of admitting fluid, but the difference is one merely of time and degree.

In our schema we can imitate pneumothorax either by thrusting a needle through trachea tube D', and puncturing the bag, or what is more convenient, by opening stop-cock B'. The result will be *instant* collapse of bag C to c, instant fall of column I' K' to the level, immediate displacement of the mediastinum, E F, to position of dotted line, with slight contraction of C, and slight falling of I K. I have already enlarged upon the clinical phenomena of pneumothorax, (page 130 *et seq.*,) they are identical with those illustrated in the schema. Although theoretically, we might conceive air to be effused into the pleura as gradually as fluid, yet practically, this is rarely the case. The escape of air, even through a small opening, is so rapid that its consequences may be rightly described as sudden. Hence in pneumothorax, as we have seen, displacement of the heart is instantaneous, and the diaphragm, no longer supported by the traction of the

lung, becomes flaccid and slightly convex downwards or upwards as the pressure of the abdominal viscera shall determine.

Any further accumulation of air produces pressure effects identical with those of fluid, and, from the greater elasticity of the element, of much greater gravity to the patient. The greatest air pressure I have ever measured in the chest has been equal to 7 inches of water; the greatest fluid pressure has been $1\frac{1}{2}$ inch of mercury⁵³: but the air-pressure proved rapidly and directly fatal, the fluid pressure symptoms being much less urgent.

The *absorption* of air from the pleura, however, when it occurs, is gradual, and in their return to the normal position, the different organs follow the same lines as in fluid effusions. In the last case related in which pneumothorax was produced by an external incision for the relief of left empyema (page 262), the heart was observed to be instantaneously displaced to the right of the sternum (it was only before situated at the left margin of the sternum). A few days later, however, the wound having closed, the heart had assumed the position it had before the operation, and the diaphragm was at this period also arched upwards. The phenomena of a limited effusion of air were then, at this period, to be observed.

I trust the above application of the schema to explain, or rather to illustrate clinical phenomena, some of which are well, others ill understood, will not be thought over-strained. I am aware that many authors would explain the phenomena differently. It may be said that atmospheric pressure has been too much left out in my descriptions. This is from no indifference to the presence of atmospheric pressure as an all-pervading power, without which none of the conditions in the chest could be for a moment maintained; but because it is the disturbing causes which, by altering the direction,

⁵³ *Med. Chir. Trans.*, vol. lix. pp. 15 and 23.

distribution, and degree of atmospheric pressure, produce the effects witnessed, and which are, therefore, the new elements and real agents at work.

There are other phenomena of chest disease which are the results of variations of the atmospheric pressure within the chest. They are of a different kind to those above referred to, and are due to obstruction to the entry of air into the lungs, whilst the powerful inspiratory muscles forcibly expanding the chest cavity create a partial vacuum there. It is for instance, rare for a patient to have a severe attack of spasmodic asthma, without suffering subsequently from œdema of the lungs and more or less bronchitis. In laryngismus and in enlarged tonsils in young children, disease of the lungs commencing with mechanical determination of blood to them, may not infrequently be observed. Part of the secondary phenomena of hooping cough may be traced to the same cause. And, entering into details which would be beyond our present object, it would not be difficult to find many other diseased conditions helped or initiated in a similar way.

INDEX.

	Page		Page
Abscess caseous of lung	103	Bronchitis, aetiology of	168
" of lung in pneumonia	209	" diagnosis	175
" pleural in pneumonia	210	" morbid appearances	172
Adhesions, pleuritic in phthisis	17	" stages of	172
Ætiology of alveolar catarrh	28	" symptoms and signs	173
" bronchitis	168	" table of symptoms	174
" phthisis	28	" treatment of	177
" pneumonia	205	" from inhaled irritants.	180
Air, access of to diseased lungs	21	" , case	182
Air-cells of lung, how first unfolded	272		
Alison, Dr. Scott, on pneumothorax	123		
Allbutt, Dr. C., on treatment of empyema	243, 244	Cachexia, in fibroid phthisis	55
Alveolar catarrh	7, 26	Carson, Dr., observations on lung tension	274
Amphoric breathing in pneumothorax	129	Case of acute tuberculo-pneumonic phthisis	85
Anæsthesia, local in paracentesis	233	" of acute empyema	248
Aphonia, functional	97	" of bronchitis from dust	182
" paralytic in phthisis	97	" of caseous abscess of the lung	112
Arsenic in hectic	109	" of catarrhal pneumonic phthisis	29
Aspiration in serious effusions	236	" of catarrhal pneumonia	38
Asthma, diagnosis from pneumothorax	138	" of emphysema	191
Asthma, a cause of bronchitis	286	" of chronic empyema	257
Astringents in diarrhoea	150	" of fibroid phthisis	49
Bacelli on vocal resonance in effusions	220	" of haemorrhagic phthisis	61
Barlow, Dr., on pleuritic effusion	242, 253	" shewing the displacement of the heart in pneumothorax	131
Bastian, Dr., on cirrhosis of the lung	48	" of recent cavity cicatrising	112
Bennett, Dr. Hughes, on local treatment of cavities	119	" of recurrent haemoptysis	69
Berkart, Dr., instrument for treatment of emphysema	197	Cases of pneumothorax tabulated	142
Blisters in tubercular laryngitis	98	" " tubercular meningitis	154—157
Blisters over cavities	116	" , tabulated 161—164	
Bowel, ulceration of in phthisis	144	Catarrh, alveolar	7, 26
Brompton Hospital records of Pneumothorax	125	Cavities in the lung	100
Bronchiectasis, causes of	19	" extension of	107
		" local treatment	119
		Cavity basic, treatment of	120
		" recent, signs of	102
		" , mode of formation	102

	Page		Page
Cavity recent extension	106	Effusions chronic pleuritic	229
" " cicatrisation	112, 115	Elastic tissue in sputa	7, 106
" quiescent	114	Elasticity of lungs, effect in respiration	16
" " closure of	115	Emetics in haemoptysis	76
" secreting	116	Emphysema	186
" " treatment of	116	" aetiology of	189
" ulcerous	118	" case related	191
" " treatment of	119	" condition of heart in	189
Chest, exploration of	24	" effect on circulation	188, 281
" regions of	24	" pathology of	187
Cheyne-Stokes breathing in meningitis	160	" phenomena of, illustrated	280
Chills in phthisis	108	" respiratory movements in	193
Clark, Dr. A., on albuminoid degeneration	56	" treatment	194
" on fibroid phthisis	9, 47	" " by rarefied and condensed air	194-195
Cœcum, ulceration of	146	Empyema	218, 239
Constipation with ulceration of bowel	149	" pressure symptoms in	225
Constitution, influence on pulmonary disease	3	" pulsating	224
Consumption, pulmonary, definition of	5	" reference to cases by Dr. Goodhart	240
" galloping	87	" secondary to pneumothorax	139
Cotton, Dr., table of physical signs	23	" treatment of	239
Cough in laryngeal phthisis	94	Enemata opiate in diarrhoea	151
" vomiting with	53	Epiglottis, ulceration of in phthisis	93
Counter-irritation in laryngeal phthisis	98	Ergot in haemoptysis	67, 76
" over cavities	116, 119	Exanthemata, bronchitis in	171
Coupland, Dr. S., drawings from sputa	105	Expectoration, rusty in phthisical pneumonia	41
Davy, Dr., analysis of gas in pneumothorax	125	" examination of	104
Diagrams from case of pneumothorax	132	" importance of	112
Diaphragm, state of in pleuritic effusion	231	Experiments of author	134
Diarrhoea in phthisis	147, 149	" of Goodwyn and Erichsen, referred to	134
" " treatment	150	Fever in phthisis	40
Diathesis, haemorrhagic	66	" " relation to signs	40
Digitalis in haemoptysis	76	Fibroid phthisis	9
Displacement of heart in pneumothorax	130	" " cachexia in	55
Dobell, Dr., on mechanical rest in phthisis	35	" " diagnosis	54-58
Donders on lung tension	274	" " pathology of	46-49
Duncan, Dr., analysis of gas in pneumothorax	126	" " physical signs	50
Dust, effect in producing catarrh	170	" " prognosis	55
Dust-bronchitis	180	" " varieties of	49
Dysenteric symptoms in phthisis	148	Flattening of chest, significance of	34
Effusions into the pleura	226	Fox, Dr. W., on chronic pneumonia	48
" " treatment of	226	Gaide, M., on displacement of heart in pneumothorax	130
" pleuritic, phenomena of, illustrated	282	Gairdner, Dr., on emphysema	190
		" on simple pneumothorax	123

	Page		Page
Gangrene of lung in pneumonia	209	Inhalations	117, 118
Garland, Dr., Experiments of	231	" in laryngeal phthisis	98
Gee, Dr., on signs of recent pleurisy	215	" in cavities	111, 118
Giant-cell	12	Intestines, ulceration of	144
Goodhart, Dr. tabulated cases of empyema	240	Jenner, Sir W. on emphysema	189
Granulations, tubercular	10	Klein, Dr. on lymphatics of the lung	9
Gravitation, effect of in hæmoptysis	64	Klebs and Mosler on effect of swallowing expectoration	147
Greenhow, Dr., on emphysema	190	Lænnec on cicatrisation of cavities	112
Hæmophilia, hæmoptysis in	66, 270	Lænnec on pneumothorax	125
Hæmoptysis, a cause of phthisis	60	" on relation of tubercle to phthisis	12
" early, temperature in	67	Laryngeal phthisis	90
" treatment of	67	Laryngitis, tubercular and syphilitic	97
" lesions secondary to	64	Laryngoscope, use of the	95
" significance of	78	Larynx, ulceration of	92
Hæmoptysis recurrent, features of	73	Legg, Dr. Wickham on hæmoptysis in hæmophilia	66
" case of	69	Louis on pneumothorax	125
" from cavities	117	Lung fibrosis of	8
" pathology of	74	" fibrous stroma	8
" recovery from	77	" parenchyma	8
" temperature in	75	Lungs, movements of	20
" treatment	76	" muscular contractility	275
Hæmoptysis spurious	265	" residual tension of	272, 273
" from epistaxis	265	Lymphatics of lungs	9, 10
" from diseased gums	266	Lymph spaces in alveolar walls	9
" hysterical	266		
" in hæmophilia	270		
" scorbutic	267		
Hæmorrhage from bowels	148		
Hayden, Dr. on displacement of heart	133		
Heart disease, a cause of bronchitis	171		
Heart in emphysema	198		
" displacement of in pleuritic effusion	221		
Heart, murmur over, in pleuritic effusion	223		
Hensley, Dr. on expansion of air-cells in infants	272		
Hereditary predisposition to tubercle	15		
Hydrothorax chronic, treatment of	238		
Inflammation, degeneration of products	5		
Inflammation in phthisis	5		
		Niemeyer on pyrexia in alveolar catarrh	21
		Night-sweats	108
		Œdema of pleura	16
		Paracentesis thoracis in serous effusion	231

	Page		Page
Paracentesis thoracis, apparatus for	232	Pneumonia	200
Parenchyma of lung	8	" adaptation of thorax in	281
Parker, Mr., on pleuritic effusion	242	" oetiology of	205
Pepper, Dr. W., on local treatment of cavities	119	" definition of	205
Percussion hints on	25	" dangers in	206, 207
Pharynx ulceration of in phthisis	94	" gangrenous	209
Phthisis pulmonalis definition of	5	" hectic fever and chills in	204
Phthisis, first stage of	21	" pleural abscess in	210
" from inhaled irritants	180	" treatment	206
" haemoptysis a cause of	60	Pneumothorax	123
" pathology of	5	" diagnosis of	137
" ulceration of bowel in	144	" displacement of heart in	130
" table of varieties	89	" nature of gas effused	125
" catarrhal-pneumonic	26	" nature of opening	126
" catarrhal, aetiology	28, 32, 181	" pathology of	123
" catarrhal, signs of arrest	34	" phenomena of illustrated	284
" catarrhal treatment	35	" side most commonly affected	125
" chronic pneumonic	44	" symptoms and signs	127
" fibroid	9, 50	" table of cases	142
" haemorrhagic	59	" treatment	140
" laryngeal	93	Pollock, Dr., on aetiology of phthisis	33
Physical examination of chest	22	Potain's aspirator	236
Pleura, diseases of the	211	Pulsating empyema	224
" oedematous	19	Pyrexia in alveolar catarrh	26
" thickening of the	18	Quinine in hectic	109
Pleurisy, simple	212	Remarks introductory	I
" " causes	212	Respiration broncho-vesicular	31
" " diagnosis	215	" Cheyne-Stokes in meningitis	160
" " pathology	212	" mechanism of	272
" " symptoms and signs	212	" in new-born infant	272
" " treatment	216	" schema	276
" local	217	" thoracic resilience in	278
" from extension	217	" tracings of in emphysema	193
" suppurative	218	" tracings of in empyema	264
" suppurative symptoms and signs	219	Rest in treatment of phthisis	36
Pleuritic adhesions	17	Rieyel on catching cold	169
" effusion, excessive signs of	221	Roberts, Dr. F. T., on treatment of pleurisy	36
" " cardiac murmur in	223	Roe, Dr. H., on paracentesis	243
" " displacement of heart	214, 221	Sanderson, Dr. Burdon on Tubercle	9
" " pulsation of fluid	224	Savin ointment counter-irritation	116
" effect of, upon pulmonary disease	122, 143		

	Page		Page
Schema of respiration	276	Treatment of empyema	
Siegle's spray-apparatus	117	" " by antiseptic	
" spray ipecacuanha	118	method	245
" spray perchloride of iron	117	" " by single opening and injection	249
Sputa, elastic tissue in	105	" " by drainage tube	254
" examination of	104	" " chronic empyema	255
Stricker on muscular tissue of lung	275	" " of pleuritic effusion	226
Sturges, Dr., on aetiology of bronchitis	169	" " of pneumonia	206
Table of bronchitis	174	Tubercle in consumption	5
" of pneumothorax	142	" development into	
" of tubercular meningitis	161	fibroid	13
" of varieties of phthisis	89	granulations	10
Temperature in early haemoptysis	67	hereditary disposition to	15
Temperature in pulmonary softening	107	nature of	10
Temperature in tubercular phthisis	82	relation to phthisis	12
Temperature in tubercular meningitis	166	seat of	10, 12
Thornton, Mr. Knowsley, on ice applications to the head	170	" and tuberculosis, distinction between	13
Thorowgood, Dr., on dry cough	37	Tubercular phthisis	80
Throat, alcoholic catarrh of	97	Tuberculation, pulmonary	80
Tissue, elastic of lung	7	Tuberculosis, miliary	82
" interstitial of lung	7	" degrees of infection producing	82
Tongue, tubercular ulcer of	94	Urine, phosphatic in tubercular meningitis	155
" in intestinal ulceration	148	Waldenburg on treatment of emphysema	197
Trachea, ulceration of	93	Waldenburg on tuberculosis after haemoptysis	60
Tracings of intra-pleural pressure	264	Walshe on Pneumothorax	125
Tracings of respiratory movements in emphysema	193	" on intestinal ulceration	149
Treatment, of emphysema	194	Williams, Dr. C. T., on hereditary disposition to phthisis	15
" of empyema	239, 262		

MEDICAL AND SURGICAL WORKS

Recently published by

H. K. LEWIS.

136 GOWER STREET LONDON, W.C.

SYDNEY RINGER, M.D.

Professor of Therapeutics at University College ; Physician to University College Hospital.

A HANDBOOK OF THERAPEUTICS. Sixth edition,
revised and enlarged, 8vo, cloth, 14s.

F. T. ROBERTS, M.D., B.Sc.

Assistant Physician and Teacher of Clinical Medicine in the University College Hospital ;
Assistant Physician, Brompton Consumption Hospital, &c.

A HANDBOOK OF THE THEORY AND PRACTICE
OF MEDICINE. Third edition, 2 vols., 8vo, cloth, 22s.

DR. V. MAGNAN.

Physician to the St. Anne Asylum, Paris, Laureate of the Institute, &c.

ON ALCOHOLISM : THE VARIOUS FORMS OF
ALCOHOLIC DELIRIUM AND THEIR TREATMENT. Translated by
W. S. GREENFIELD, M.D., M.R.C.S. 8vo, cloth, 7s 6d.

W. SPENCER WATSON, F.R.C.S. ENG., B.M. LOND.

Surgeon to the Great Northern Hospital ; Surgeon to the Royal South London Ophthalmic
Hospital, and to the Central London Ophthalmic Hospital ; Formerly Assistant
Surgeon to King's College Hospital.

DISEASES OF THE NOSE AND ITS ACCESSORY
CAVITIES. Profusely illustrated, demy 8vo, cloth, 18s.

WILLIAM A. HAMMOND, M.D.

Professor of Mental and Nervous Diseases in the Bellevue Hospital Medical College,
New York, &c.

A TREATISE ON THE DISEASES OF THE NER-
VOUS SYSTEM. Sixth edition, rewritten and enlarged, with 109 illustra-
tions, large 8vo, cloth, 25s.

HORACE DOBELL, M.D.

Consulting Physician to the Royal Hospital for Diseases of the Chest, &c., &c.

ON DIET AND REGIMENT IN SICKNESS AND HEALTH, and on the Interdependence and Prevention of Diseases and the Diminution of their Fatality. Sixth revised and enlarged édition, small 8vo, cloth, 6s.

By the same Author.

AFFECTIONS OF THE HEART, AND IN ITS NEIGHBOURHOOD. Cases, Aphorisms, and Commentaries. Illustrated by the Heliotype process. 8vo, cloth, 6s 6d.

AUSTIN FLINT, JUN., M.D.

Professor of Physiology and Physiological Anatomy in the Bellevue Medical College, New York; Attending Physician to the Bellevue Hospital; Consulting Physician for the Class of Nervous Diseases to the Bureau of Medical and Surgical Relief for Out-door Poor, Bellevue Hospital; Fellow of the New York Academy of Medicine; Fellow of the Medical Society of the County of New York, &c.

A TEXTBOOK OF HUMAN PHYSIOLOGY; DESIGNED FOR THE USE OF PRACTITIONERS AND STUDENTS OF MEDICINE. Large 8vo, cloth, copiously illustrated by plates and wood engravings, £1 8s.

J. WICKHAM LEGG, M.D.

Fellow of the Royal College of Physicians and of the Society of Antiquaries of London; Demonstrator of Morbid Anatomy in St. Bartholomew's Hospital.

A GUIDE TO THE EXAMINATION OF THE URINE; INTENDED CHIEFLY FOR CLINICAL CLERKS AND STUDENTS. Fourth edition, feap. 8vo, cloth, 2s 6d.

By the same Author.

A TREATISE ON HÆMOPHILIA, SOMETIMES CALLED THE HEREDITARY HÆMORRHAGIC DIATHESIS. Feap. 4to, cloth, 7s 6d.

J. F. MEIGS, M.D.,

Lecturer on Clinical Medicine in the University of Pennsylvania.

AND

W. PEPPER, M.D.,

Consulting Physician to the Children's Hospital, Philadelphia.

A PRACTICAL TREATISE ON DISEASES OF CHILDREN. Sixth edition, revised and enlarged, roy. 8vo, cloth, 28s.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. OXON.
Professor of Surgery in the Jefferson Medical College of Philadelphia.

- A PRACTICAL TREATISE ON THE DISEASES, INJURIES, AND MALFORMATIONS OF THE URINARY BLADDER, THE PROSTATE GLAND, AND THE URETHRA. Third Edition, revised and edited by S. W. GROSS, A.M., M.D., Surgeon to the Philadelphia Hospital. Illustrated by 170 engravings, 8vo, cloth, 18s.
-

DR. FELIX VON NIEMEYER.

- A TEXTBOOK OF PRACTICAL MEDICINE, WITH PARTICULAR REFERENCE TO PHYSIOLOGY AND PATHOLOGICAL ANATOMY. Translated from the eighth German edition, by special permission of the Author, by GEORGE H. HUMPHREY, M.D., and CHARLES E. HACKLEY, M.D. 2 vols., large 8vo, cloth, 36s.
-

GURDON BUCK, M.D.

- CONTRIBUTIONS TO REPARATIVE SURGERY : Showing its Application to the Treatment of Deformities, produced by Destructive Disease or Injury; Congenital Defects from Arrest or Excess of Development; and Cicatrical Contractions from Burns. Illustrated by numerous engravings. Large 8vo, cloth, 9s.
-

CHARLES HENRY RALFE, M.A., M.D., &c.
Physician to the Seaman's Hospital; Lecturer on Physiological Chemistry at St. George's Hospital.

- OUTLINES OF PHYSIOLOGICAL CHEMISTRY ; INCLUDING THE QUALITATIVE AND QUANTITATIVE ANALYSIS OF THE TISSUES, FLUIDS, AND EXCRETORY PRODUCTS. Fcap. 8vo, cloth, 6s.
-

J. ASHBURTON THOMPSON.

Surgeon at King's Cross to the Great Northern Railway Company, Surgeon Accoucheur to the Royal Maternity Charity, &c.

- FREE PHOSPHORUS IN MEDICINE WITH SPECIAL REFERENCE TO ITS USE IN NEURALGIA. A Contribution to Materia Medica and Therapeutics An account of the History, Pharmaceutical Preparations, Dose, Internal Administration, and Therapeutic uses of Phosphorus: with a complete Bibliography of this subject, referring to nearly 200 works upon it. Demy 8vo. cloth, pp. 275, 7s 6d.
-

J. THOMPSON DICKSON, M.A., M.B. CANTAB.

Lecturer on Mental Diseases at Guy's Hospital: late Superintendent of St. Luke's Hospital.

- THE SCIENCE AND PRACTICE OF MEDICINE IN RELATION TO MIND, the Pathology of the Nerve-Centres, and the Jurisprudence of Insanity, being a Course of Lectures delivered at Guy's Hospital. Illustrations in chromo-lithography, and physiological portraits, 8vo, cloth, 14s.

JOHN S. PARRY, M.D.

Obstetrician to the Philadelphia Hospital, to the Department for the Diseases of Women in the Presbyterian Hospital, Fellow of the College of Physicians of Philadelphia, Vice-President of the Obstetrical and Pathological Societies of Philadelphia, &c.

EXTRA-UTERINE PREGNANCY; ITS CAUSES, SPECIES, PATHOLOGICAL ANATOMY, CLINICAL HISTORY, DIAGNOSIS, PROGNOSIS AND TREATMENT. 8vo, cloth, 8s.

E. RANDOLPH PEASLEE, M.D., LL.D.

Professor of Gynaecology in the Medical Department of Dartmouth College; Attending Surgeon of the New York State Woman's Hospital; President of the New York Academy of Medicine, &c., &c.

OVARIAN TUMOURS: THEIR PATHOLOGY, DIAGNOSIS AND TREATMENT, ESPECIALLY BY OVARIOTOMY. Illustrations, roy. 8vo, cloth, 16s.

F. HOFFMAN, PH.D.

Pharmacist in New York.

MANUAL OF CHEMICAL ANALYSIS AS APPLIED TO THE EXAMINATION OF MEDICINAL CHEMICALS. A Guide for the determination of their Identity and Quality, and for the Detection of Impurities and Adulterations. For the use of Pharmacists, Physicians, Druggists and Manufacturing Chemists, and of Pharmaceutical and Medical Students. Royal 8vo, cloth, 12s.

G. M. BEARD, A.M., M.D.

Fellow of the New York Academy of Medicine; and

A. D. ROCKWELL, A.M., M.D.

Fellow of the New York Academy of Medicine.

A PRACTICAL TREATISE ON THE MEDICAL AND SURGICAL USES OF ELECTRICITY. Including Localized and General Faradization; Localized and Central Galvanization; Electrolysis and Galvano-Cautery. Second edition, revised, enlarged, and mostly re-written. With nearly 200 illustrations, royal 8vo, cloth, £1 5s.

D. F. WINKEL.

Formerly Professor and Director of the Gynaecological Clinic at the University of Rostock.

THE PATHOLOGY AND TREATMENT OF CHILD-BED: A Treatise for Physicians and Students. Translated from the Second German Edition, with many additional notes by the Author, by J. B. CHADWICK, M.D. 8vo, cloth, 14s.

P. CAZEAUX.

Member of the Imperial Academy of Medicine; Adjunct Professor in the Faculty of Medicine of Paris, &c.

A THEORETICAL AND PRACTICAL TREATISE ON MIDWIFERY, INCLUDING THE DISEASES OF PREGNANCY AND PARTURITION. Revised and Annotated by S. TARNIER. Translated from the seventh French edition by W. R. BULLOCK, M.D. Royal 8vo, cloth, over 1100 pages, 175 illustrations, 30s.

Ja

